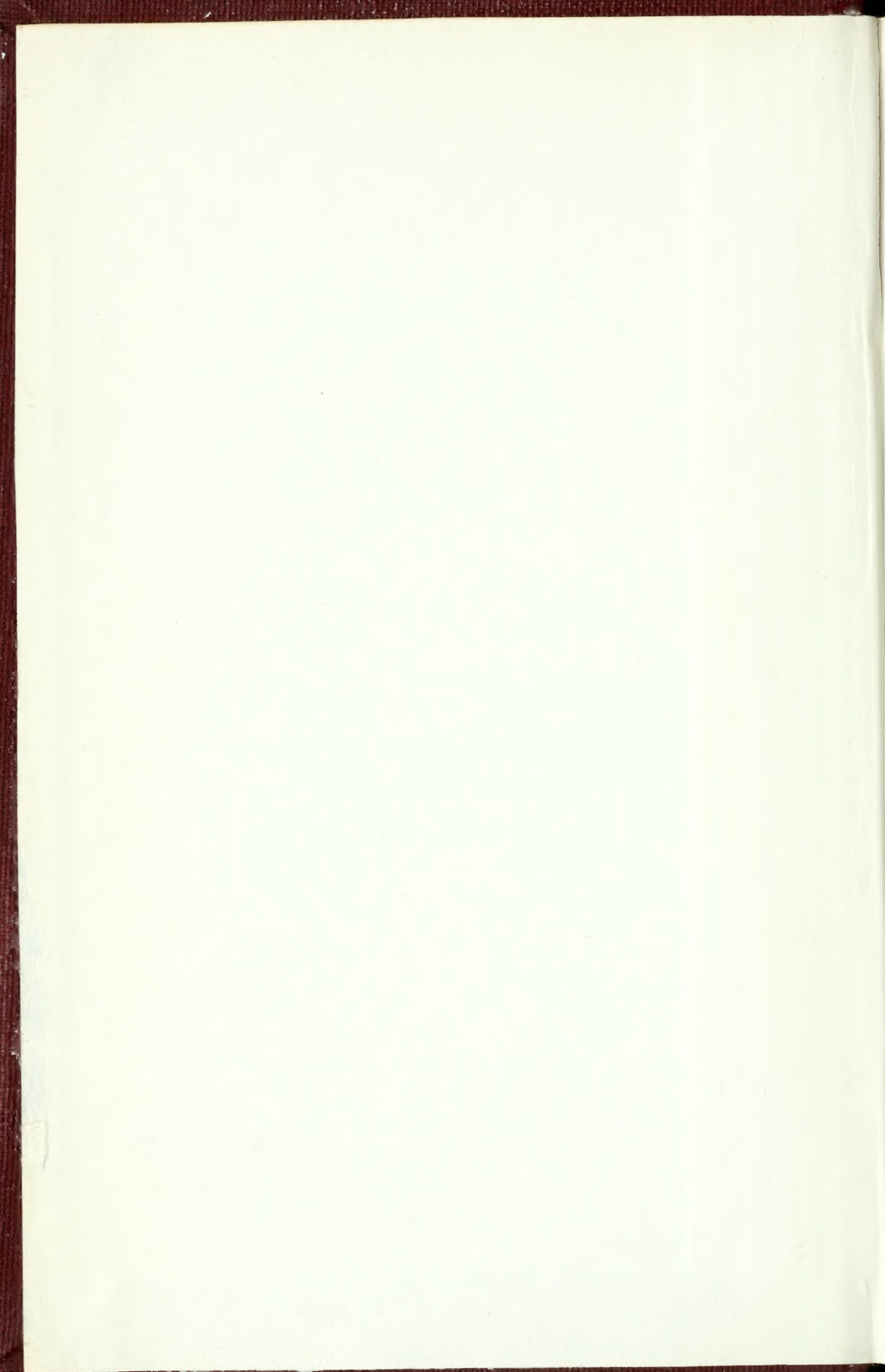




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Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING
THE INDEX OF OTOLARYNGOLOGY

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TABLE OF CONTENTS.

I.—The Combined Intranasal and External Operation on the Lacrimal Sac. (Mosher-Toti.) By HARRIS P. MOSHER, M. D., Boston.....	1
II.—Dacryorhinocystotomy; Combined Methods. By W. E. SAUER, M. D., St. Louis.....	25
III.—Study of Reinforcement of Sound by Means of the Schaefer Resonators. By JOHN P. MINTON, PH. D., and ROBERT SONNENSCHN, M. D., Chicago.....	45
IV.—Regional Anatomy of the Tear Sac. By JAS. M. PATTON, M. D., F. A. C. S., Omaha.....	58
V.—Indications, Contraindications and Preparation for Dacryocystorhinostomy. By DR. RALPH A. FENTON, Portland, Oregon.....	67
VI.—Use of Tuning Fork Stem for Both Air and Bone Conduction in the Rinne Test. By ROBERT SONNENSCHN, M. D., and JOHN P. MINTON, PH. D., Chicago.....	85
VII.—Aural Exostosis. By FRANK A. BURTON, M. D., Denver, Colo.....	97
VIII.—Consideration of the Radical Mastoid. By C. M. SAUTTER, M. D., New York.....	118
IX.—Case of Polyarthrits Complicating Mastoiditis. Operation. Recovery. By DUNBAR ROY, A. M., M.D., Atlanta.....	129
X.—The End Results of Radical Operations on the Accessory Sinuses. By ROSS HALL SKILLERN, Philadelphia.....	139
XI.—Treatment of Otitis Media With Tuberculin. By G. THOMAS VON COLDITZ, M. D., Chicago.....	149
XII.—The Practical Diagnostic Value of Tests of the Vestibular Mechanism. By FRANK L. DENNIS, M. D., Colorado Springs.....	160
XIII.—A Simple Knot for Intranasal Sutures. By WILSON JOHNSTON, M. D., Portland, Oregon.....	201
XIV.—Compensatory Nasal Growths. By J. A. PRATT, M. D., F. A. C. S., Minneapolis.....	211
XV.—Report of a Case of Tonsillar Cyst Which Protruded From the Tonsil as a Pedunculated Tumor. By ARTHUR W. PROETZ, M. D., St. Louis.....	223
XVI.—Report of a Case of Codein Poisoning. By ARTHUR W. PROETZ, M. D., St. Louis.....	231

XVII.—Report of Two Cases of Salivary Calculus. By ARTHUR W. PROETZ, M. D., St. Louis.....	233
XVIII.—Traumatic Facial Diplegia, With Involvement of the Sixth Nerves, a Portion of the Left Third Nerve, Also the Fifth and Eighth Nerves, With Dislocation of the Atlas, Etc. By GEORGE F. KEIPER, A. M., M. D., Lafayette, Indiana.....	236
XIX.—Two Cases of Empyema of the Maxillary Sinus of Dental Origin. Alveolar Fistula. Radical and Plastic Operation. By J. L. MAYBAUM, M. D., F. A. C. S., New York.....	273
XX.—Chronic Suppurative Otitis Media. By HERRMANN E. BOZER, M. D., Rochester, Minnesota.....	277
XXI.—Complications of Paranasal Sinus Disease in Infants and Young Children. By L. W. DEAN, M. D., Iowa City.....	285
XXII.—Observations on the Art and Technic of Bronchoscopy and Esophagoscopy. By THOMAS HUBBARD, M. D., Toledo.....	298
XXIII.—The Radical Frontal Sinus Operation, With Report of Cases. By FIELDING O. LEWIS, M. D., Philadelphia.....	305
XXIV.—Depressed Nasal Deformities. A Comparison of the Prosthetic Values of Paraffin, Bone, Cartilage and Celluloid, With Report of Cases Corrected With Celluloid Implants by the Author's Method. By JOSEPH D. LEWIS, M. D., Surgeon-in-Chief, Department of Eye, Ear, Nose and Throat, Minneapolis General Hospital, Minneapolis.....	321
XXV.—The Relation Between Thyrotoxicosis and Tonsillar Infection. By LOUIS E. BROWN, M. D., Akron, Ohio.....	367
XXVI.—Submucous Resection—Complications and After-Results. By N. S. WEINBERGER, M. D., Robert Packer Hospital, Sayre, Pa.....	387
XXVII.—Treatment of Acute Sinus Infections. By LEE M. HURD, M. D., New York.....	394
XXVIII.—Some Further Observations on the Etiology and Treatment of Maxillary Sinusitis. By HOWARD V. DUTROW, M. D., Dayton, Ohio.....	398
XXIX.—The Upper Respiratory Tract in Granite Dust Inhalation. By D. C. JARVIS, M. D., Barre, Vt.....	405
XXX.—Pulmonary Abscess Following Nose and Throat Surgery. By JOSEPH PRENN, M. D., Boston.....	413
XXXI.—Infection of the Accessory Sinuses in Children, With Report of Cases. By E. A. LOOPER, M. D., Baltimore.....	417

XXXII.—Some Remarks on Nystagmus. By G. W. MACKENZIE, M. D., Philadelphia.....	427
XXXIII.—The Diagnosis and Treatment of Pulmonary Conditions Through the Bronchoscope. From the Medical School, Creighton University, Department of Otolaryngology. By B. M. KULLY, M. D., Omaha.....	437
XXXIV.—The Structure and Function of the Crista Ampullaris. By GEORGE E. SHAMBAUGH, M. D., Chicago.....	443
XXXV.—A New Magnifying Glass for Otoscopy. By ALEXANDER ZEBROWSKI, M. D., New York.....	453
XXXVI.—Otitis Media Complicating Operations on the Gasserian Ganglion. By H. R. LYONS, M. D., Rochester, Minnesota	457
XXXVII.—Nasal Reflex. By ROBERT F. RIDPATH, M. D., Philadelphia	464
XXXVIII.—Tumors of the Jaw From the Standpoint of the Rhinologist. By J. D. WHITHAM, M. D., New York.....	474
XXXIX.—Phlebothrombosis of the Intracranial Sinuses. By C. COULTER CHARLTON, M. D., Atlantic City, N. J.....	485
XL.—Dental Relations of the Eye, Ear, Nose and Throat. By W. H. HASKIN, M. D., New York.....	497
XLI.—The Correction of External Deformities of the Nose. By SIDNEY ISRAEL, M. D., Houston, Texas.....	504
XLII.—A Case of Congenital Hemimacroglossia, With Disturbance of the Locomotor Apparatus of the Side Opposite the Lingual Lesion. By J. N. ROY, M. D., Associate Professor at the University of Montreal; Laureate of the Academy of Medicine of France, Montreal.....	519
XLIII.—Sinusitis From Swimming. By RALPH A. FENTON, A. B., M. D., Portland, Oregon.....	526
XLIV.—The Effect of Pressure Changes in the External Auditory Canal on Acuity of Hearing. By AUGUSTUS G. POHLMAN, M. D., Department of Anatomy, St. Louis University, and FREDERICK W. KRANZ, PH. D., Wallace Clement Sabine Laboratory of Acoustics, Riverbank Laboratories, Geneva, Ill.....	545
XLV.—Idiopathic Perforation of the Nasal Septum. Autoplasty With a Pedunculated Flap of Mucous Membrane. Cure. By J. N. ROY, M. D., Associate Professor at the University of Montreal	554

XLVI.—Otitis Media, Mastoiditis and Disease of Nasal Accessory Sinuses as Causative Factor in Malnutrition in Children. By T. H. ODENEAL, M. D., Ophthalmologist and Otolaryngologist to the Beverly Hospital and Massachusetts State Infirmary, Beverly, Mass.....	561
XLVII.—Relations Between the Eye and Ear (Including the Vestibular Organ). By PROF. J. VAN DER HOEVE, Leiden, Holland	571
XLVIII.—Metastatic Laryngeal Arthritis. By S. O. FIELDS, M. D., Norfolk	588
XLIX.—Further Observations of Mastoid Structure by Means of the X-Ray. By NOLTON BIGELOW, M. D., and ISAAC GERBER, M. D., Providence, R. I.....	641
L.—The Incidence and Histopathology of Bone and Cartilage in the Tonsil. By CARL VERNON WELLER, M. S., M. D., Associate Professor of Pathology, University of Michigan, Ann Arbor.....	687
LI.—Note on the Relation of the Optic Nerve to the Last Posterior Ethmoid Cell. By S. B. WESTLAKE, M. D., St. Louis	729
LII.—A Study of the Mechanism of Pain as Seen in Rhinologic and Laryngologic Cases. By L. Hubert, M. D., Assistant Surgeon, Manhattan Eye, Ear and Throat Hospital, New York	749
LIII.—Recent Nasal Fractures. By IRA FRANK, M. D., Chicago	768
LIV.—The Otologist in the Field of Sociology (the Alliance of Medical and Social Sciences in the Prevention and Palliation of Acquired Deafness). By WENDELL C. PHILLIPS, M. D., New York.....	780
LV.—Aids to Hearing. By GORDON BERRY, M. D., Worcester, Mass.....	807
LVI.—Mastoiditis in the Aged. By RALPH A. FENTON, A. B., M. D., Portland, Ore.....	820
LVII.—Alcohol Injections as a Possible Adjunct to Tonsillectomy Under Local Anesthesia. By ROBERT SONNENSCHN, M. D., Chicago.....	827
LVIII.—The Clinical Diagnosis of Laryngeal Tuberculosis. By FRANK R. SPENCER, A. B., M. D., F. A. C. S., Boulder, Colorado	838

LIX.—The Differentiation of Neurolabyrinthitis Syphilitica and Labyrinthitis Syphilitica Tarda. By GEORGE W. MACKENZIE, M. D., Philadelphia	847
LX.—Parotid Fistulæ After Mastoid Operation. By W. E. GROVE, M. D., F. A. C. S., Milwaukee.....	855
LXI.—Lymphoid Tissue in the Tonsillar Fossæ Following Tonsillectomy. By B. B. GRICHTER, M. D., and S. J. PEARLMAN, M. D., Chicago.....	860
LXII.—Clinical and Pathologic Study of Tonsil Cases. By AUSTIN G. MORRIS, M. D., Rochester, N. Y.....	864
LXIII.—Radical Mastoidectomy; Complete Healing; Method of Controlling Persistent Vertigo. By J. L. MAYBAUM, M. D., New York.....	875
LXIV.—Some of the Aspects of Aphonia, Psychic and Otherwise. By JAMES A. BABBETT, M. D., Philadelphia.....	879
LXV.—Foreign Bodies in Upper Air and Food Passages—A Clinical Report. By HARRINGTON B. GRAHAM, M. D., San Francisco	886
LXVI.—The Cochlea-Palpebral Reflex in Infants. By THEODOR DEMETRIADES, M. D., Assistant on the Ear, Nose and Throat Department of the University Policlinic, Athens, Greece	894
LXVII.—Precautions in Using Local Anesthesia—Report of a Death. By CURTIS C. EVES, M. D., Philadelphia.....	904
LXVIII.—The Waring Suction Tonsillectomy. By J. B. H. WARING, M. D., Blanchester, Ohio	913
LXIX.—Otogenic Sympathetic Meningitis—Report of a Case With Recovery After a Radical Mastoid Operation. By JESSE WRIGHT DOWNEY, JR., M. D., F. A., PIACENZA, M. D., and OSCAR G. COSTA, M. D., House Staff, Mercy Hospital, Baltimore	923
LXX.—Contribution to the Pathology of the Labyrinth. By J. S. FRASER, M. B., CH. B., F. R. C. S. Ed., Edinburgh.....	953
LXXI.—Observations of Adenoids Removed With a Direct Vision Adenotome. By I. D. KELLEY, JR., M. D., St. Louis	1053
LXXII.—A Case of Epithelioma of the Esophagus With Some Unusual Features—Specimen. By SAMUEL MCCULLAGH, M. D., New York.....	1083

LXXIII.—Surgical Diathermy by the Needle Method. By ALFRED LEWY, M. D., Chicago.....	1086
LXXIV.—Further Studies of Fatalities Following Operations on the Nose and Throat. By H. W. LOEB, M. D., St. Louis	1103
LXXV.—The Anatomy of the Sphenoid Fissure. By GREENFIELD SLUDER, M. D., St. Louis.....	1108
LXXVI.—Deafness as a Sequela to Mumps. By GEORGE H. WILLCUTT, M. D., San Francisco.....	1121
LXXVII.—Congenital Atresia of the Postnasal Orifices. By JAMES E. LEBENSOHN, M. S., M. D., Chicago.....	1128
LXXVIII.—Nontraumatic Hernia of the Diaphragm—An Embryologic Viewpoint. By LYMAN G. RICHARDS, M. D., Boston	1145
LXXIX.—Radical Operations on the Maxillary Sinus and Damage to the Teeth. By ROBERT H. IVY, M. D., D. D. S., Philadelphia	1197
LXXX.—Otitic Cholesteatomata. By S. MACCUEN SMITH, M. D., Philadelphia	1203
LXXXI.—A Survey of the Hay Fever Question—A Critical Review of the Situation Relative to the Etiology and Treatment. By OTTO J. STEIN, M. D., Chicago.....	1214
LXXXII.—Brain Abscess of Otitic Origin in the Left Temporo-Occipital Region With All the Typical Symptoms. Operation. Recovery. By ALEXANDER ZEBROWSKI, M. D., New York	1224
LXXXIII.—Some General Effects of Local Anesthetics Administered as in Tonsillectomy. By ELLISON L. ROSS, PH. D., M. D., Chicago.....	1229
LXXXIV.—Tumor of the Carotid Gland With Stokes-Adams Syndrome. By G. W. BOOT, M. D., Chicago.....	1241
LXXXV.—Klebs-Loeffler Infection of the Middle Ear and Mastoid. By RUSSELL WEBBER, M. D., Waterbury, Conn.....	1246
SOCIETY PROCEEDINGS.....	316, 595, 926, 1251
Chicago Laryngological and Otological Society.....	316, 595, 606, 635, 926, 934, 1251
Meeting of the Section of Otolaryngology of the College of Physicians, Philadelphia, December 20, 1922.....	1259

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INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

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No. 1.

I.

THE COMBINED INTRANASAL AND EXTERNAL
OPERATION ON THE LACRIMAL SAC.*
(MOSHER-TOTI.)

BY HARRIS P. MOSHER, M. D.,

BOSTON.

My job, before I take the count, is to champion the combined intranasal and external operation on the tear sac. I shall first give the results of a series of combined operations and state the conclusions drawn from a study of the cases, then in order review briefly the applied anatomy of the operation, and describe the steps of the procedure. In closing I shall refer to certain important and special points connected with the operation. Also I shall distribute a few bouquets, mostly to myself, and metaphorically send a few flowers to my opponents.

RESULTS AND CONCLUSIONS.

During the past two years seventy-one cases of dachryocystitis have been operated by the combined intranasal and ex-

*Illustrations by the writer.

ternal operation. Forty-two (42) patients recently reported in answer to a follow-up letter, the purpose of which was to give an opportunity to check up end results. It was found in the review of the cases that the pus was cured in over ninety per cent of the patients, and the epiphora in seventy-five.

In the first cases of the series the skin incision healed as a disfiguring scar. Experience has shown that the incision should not be placed too near the inner canthus. At first the distance was 6 mm. or less, and it was made on a curve throughout. It is now placed at least 12 mm. from the inner canthus, and is made nearly straight. At present a flat, invisible scar is the rule. Experience has shown that the anterior end of the middle turbinate should be removed in all cases and the anterior ethmoidal cells adjacent to the inner sac wall curetted if a lasting result is to be obtained. It has been found equally important to correct a high obstructing deviation of the septum. Three cases in which this was not done were better for a while, then relapsed. They were taken into the hospital and the septal deformity corrected.

Before operating on the sac stricture of the canaliculus should be excluded. Two partial failures in the series of combined operations were due to overlooking this condition.

THE APPLIED ANATOMY OF THE LACRIMAL SAC.

The posterior half of the bed of the sac is made by the thin lacrimal bone, the anterior half by the stout posterior edge of the ascending process of the superior maxilla. It is easy to enter the nose through the posterior half of the bed of the sac. One of the anterior ethmoidal cells, the lacrimal or the unciform cell, overlaps a part of the bed of the sac. This cell varies greatly in size. In series with the lacrimal cell and in front of it a second anterior ethmoidal cell—the agger nasi—is often found. This overlies the inner surface of the ascending process of the superior maxilla over the prominence of the process called the agger nasi. In entering the nose from the orbit by way of the bed of the sac one or both of the ethmoidal cells just mentioned must be opened. In approaching the sac intranasally the same cells are encountered. Owing to the thickness of the ascending process of the maxilla in front of the sac the intranasal approach to the sac is round a corner or through the thickest part of the ascending process.

The tip of the middle turbinate overlies the unciform cell, and a part or the whole of the agger nasi cell when this is present. In order, therefore, to get a free opening into the nose opposite the lacrimal sac it is necessary to remove the tip of the middle turbinate and the lacrimal and the agger-nasi cells.

The inner bony wall of the nasal duct has the same two components as the bed of the sac. It is hard to open the nasal duct thoroughly from the nose, but easy to do from the orbit.

THE COMBINED OPERATION ; INTRANASAL PROCEDURES.

The intranasal part of the operation consists in removing the anterior end of the middle turbinate and in curetting the anterior ethmoidal cells.

The removal of the tip of the turbinate can be done a few days before the operation. This gives somewhat less bleeding at the operation on the sac. If the operator prefers, the curetting of the anterior ethmoidal cells can be done later through the opening made into the nose in the third step of the operation. The removal of the middle turbinate tip also can be done at this time, thus making the whole operation on the sac an external operation done wholly by sight.

In whatever way these preliminary procedures are carried out, the usual care should be taken not to wound the mucous membrane of the septum for fear of subsequent nasal adhesions.

Experience has shown that it should be made a routine to remove the tip of the middle turbinate, and the overlapping anterior ethmoidal cells. An obstructing high deviation of the septum should be corrected before the operation on the sac if a lasting result is to be secured.

THE COMBINED OPERATION ; EXTERNAL PROCEDURES.

The First Step. The Incision.—The incision is placed half way between the inner canthus of the eye and the edge of the bridge of the nose. It begins on a level with the summit of the globe and is carried downward in a nearly straight line a centimeter and a half to two centimeters. It is carried to the bone throughout its whole length. With a flat chisel the subcutaneous tissues, the internal palpebral ligament and the peri-

osteum are separated from the ascending process of the superior maxilla. The subcutaneous tissues and the periosteum over the nasal bone are undisturbed.

Bleeding is controlled by placing metal clips on the edges of the incision. It was found that selfretaining retractors get in the way. The incision is held open by forceps or by small double hooks.

In order to get a flat invisible scar my experience to date may be summarized as follows:

1. Make the incision at least 12 mm. from the inner canthus.
2. Make the incision practically straight.
3. The length of the incision varies between $1\frac{1}{2}$ and $2\frac{1}{2}$ cm.
4. Do not elevate the subcutaneous tissues and the periosteum from the nasal bone.
5. Do not bring the bone opening so far forward that the skin incision lies over it.
6. Suture the anterior edge of the outer or remaining sac wall to the periosteum or the subcutaneous tissues over the nasal bone.
7. In closing the incision take time enough and put in enough sutures to get good coaptation.

The Second Step. Turning the Sac Out of Its Bed.—The sac is turned out of its bed by entering the orbit with the flat chisel above the sac and working downward and backward. The elevation of the periosteum from the inner wall of the orbit is carried a little beyond the crest of the lacrimal bone. This allows the sac to be turned well outward. Inferiorly the beginning of the nasal duct should be exposed and the surface of the ascending process of the superior maxilla opposite the beginning of the duct.

The Third Step. Making the Nasal Opening.—The third step consists in making an opening into the nose opposite the lacrimal sac the size of the sac. The opening is started by forcing a Citelli punch through the posterior part of the bed of the sac because the posterior half of the bed of the sac is made by the thin lacrimal bone. The opening is enlarged upward to the top of the bed of the sac, and backward to the crest of the lacrimal bone. Anteriorly

a little of the posterior edge of the ascending process of the superior maxilla is bitten away. Usually the Citelli punch is not strong enough to cut the ascending process of the superior maxilla, and it must be replaced by a large Kerrison punch.

In making the opening into the nose the nasal mucous membrane is trimmed flush with the bone opening. The anterior end of the middle turbinate is removed to the posterior rim of the opening. Any anterior ethmoidal cells which overlap the nasal opening are similarly dealt with.

In removing or trimming the anterior end of the middle turbinate the conchotome or ring punch can be introduced from below upward through the nose and watched through the nasal opening or introduced directly into the nasal opening.

The Fourth Step. The Removal of the Inner Half of the Sac.—The fourth step consists in removing the inner half of the sac. This is best accomplished by making a perpendicular incision along the full length of the anterior edge of the sac. From the top of this incision, and making a right angle with it, a second incision is carried horizontally backward across the summit of the sac. The incisions can be made entirely with a knife, or started with a knife and finished with scissors. The two incisions allow the inner wall of the sac to be turned inward and backward as a single flap. By steadying this with forceps it is easy to cut downward paralleling the posterior edge of the inner wall of the sac, and to remove it as one piece.

If the two walls of the sac are adherent a probe passed through the canaliculus into the sac is a great help in starting the first incision.

The Fifth Step. Removal of the Inner Wall of the Nasal Duct.—The fifth step consists in the removal of the inner bony wall of the nasal duct down to the upper edge of the inferior turbinate. This is accomplished by straddling the inner wall of the duct with a small conchotome and biting downward. This removes a part of the inner bony wall of the duct but leaves a sharp spur which is the anterior portion of the inner wall of the duct projecting backward from the posterior surface of the ascending process of the superior maxilla. The spur is removed by downward strokes of a chisel. After the removal of the spur the mem-

branous nasal duct is widely exposed. The inner wall of the membranous nasal duct is now removed with scissors or conchotome.

The Sixth Step. The Closing of the Wound.—If there has been much bleeding from the ethmoidal region the upper part of the nose is lightly packed with a narrow strip of gauze liberally smeared with vaseline. Otherwise no packing is used. In the first operations the incision was closed with fine silk sutures, the eye cleansed, the lids anointed and the eye closed and covered with a pressure bandage. Before closing the incision I now suture the anterior edge or the remaining or outer wall of the sac to the cut edge of the periosteum over the nasal bone. One or two sutures of small plain catgut are used for this purpose. This is done on the theory that it prevents sagging of the subcutaneous tissues and makes a better scar.

The nasal packing is removed in twelve to twenty-four hours. If there is much edema after the operation the lower part of the incision is opened with a probe and the pressure bandage is renewed daily for three or four days after cleaning the cornea and anointing the lids. If there is little edema the bandage is discarded in forty-eight hours. If the incision suppurates the lower part is opened, the bandage left off, the eye cleaned frequently, and hot applications applied. A suppurating incision usually becomes clean in a week or ten days.

HOW THE COMBINED OPERATION DIFFERS FROM THE TOTI OPERATION.

In the combined operation the anterior end of the middle turbinate and the overlapping anterior ethmoidal cells are removed as a routine. Toti practiced these procedures only when he felt that they were necessary. The correction of a high deviation of the septum is believed necessary as a preliminary to the combined operation. Submucous resection of the septum was not extensively practiced, if at all, when Toti published his operation.

In the combined operation the first opening into the nose is made through the thin lacrimal bone, not by attacking the ascending process. Toti saves the nasal mucous membrane

until the bone opening is completed. In the combined operation no attempt is made to save it, but instead it is removed along with the bone and then trimmed flush with the bone opening. The bony internal wall of the nasal duct and the inner membranous wall of the duct are removed down to the upper edge of the inferior turbinate.

REMARKS ON THE COMBINED OPERATION.

Crusting of the Middle Meatus.—Crusting in the middle meatus after the combined operation has caused many a solemn nodding of the head. Experience has shown that the fears on this score are groundless. Blowing of nasal secretion into the eye, another obsession of some ophthalmologists, has not occurred.

The Scar.—One keloid of the scar—which is disappearing under X-ray—and three or four bowstring scars have resulted in seventy cases of the combined operation. They occurred in the first operations in which the incision was made curved throughout and too near the inner canthus.

Probing After the Combined Operation.—Probing was not used in the after treatment. There is no law against using the probe should it be indicated. Patients, of course, shun it. One should stand ready to use the probe whenever it is indicated. The follow up shows that nine (9) cases need probing.

A Slit Canaliculus.—In order to get a cure of epiphora, one would naturally suppose that both puncta and both canaliculi would have to be intact and functioning. This is not necessarily the case. One patient, a boy of twelve, had the lower punctum sealed and pus coming only out of the upper one. This boy, however, had a perfect result in the cure of pus and epiphora. One patient who was operated for recurrent abscesses had laking in the inner canthus and but little epiphora. The lower canaliculus was permanently slit for 3 mm. To date—four months—she remains cured of the pus and the recurring abscesses, yet the laking and the epiphora remain the same. On the other hand, a woman in the sixties had the lower canaliculus permanently slit for what appeared to be its whole length. She, however, is entirely cured of the epiphora. These cases would seem to show that if the lower canaliculus is slit for a part or the whole of its course one

cannot predict what the result of the operation will be, as far as the epiphora is concerned.

The Use of the Silver Wire Style.—In cases which have been neglected, that is, cases which have come to a permanent fistula, and in cases which have been hacked at and the sac partially removed—and there are many such—one or both of the canaliculi may be closed and the common punctum sealed. In such cases it is possible to obtain an occasional success by doing the combined operation, forcing open one or both canaliculi and the common punctum and introducing a style of silver wire and leaving it some six weeks.

The Hydraulics of the Sac.—Our conception of the hydraulics of the sac have been upset by the success of the intranasal and the external operations. The long arm of the syphon which sounds so well from the standpoint of physiology is surgically done away with in these operations but still the sac drains. I have wondered whether the passage of the air through the inferior meatus in the normal case does not by suction help to empty the nasal duct and the sac. It is partly on this theory that I insist on making a new meatus opposite the common punctum of the sac so that the air can freely circulate there. In one case in which the lower punctum was sealed the upper punctum carried on adequate drainage after the combined operation.

TWO SIDE REMARKS.

Probing of the Nasal Duct.—Probing of the nasal duct is a natural procedure to try. It will occasionally succeed in the less severe cases such as those usually met with in private practice. Hospital cases are of another type and probing in these cases has not a good record.

Rapid dilatation of the nasal duct is an operation which will succeed at times. It easily becomes brutal. This operation raises its head every five or ten years. Probably it will do so periodically until the millennium.

Extirpation of the Sac.—Extirpation of the sac is admittedly an illogic, not a physiologic operation. It will, of course, cure the pus, but Gilbert's figures from the Massachusetts Charitable Eye and Ear Infirmary show that in fifty per cent of the cases so operated the eye remains disabled by persis-

tent epiphora. Any frank set of figures—not to use the harsher word—I believe will show the same result. Extirpation should be reserved for the old and feeble.

THE FOLLOW UP.

Three weeks ago, in order to check up the results to date of the operated cases, the patients were asked to report at the hospital. Forty (40) patients reported in two (2) groups of twenty (20). Two (2) private cases were personally seen and added to the list. The first and so the older cases came in the first group. The time that had elapsed since the cases were operated varied from nine (9) months to two (2) years for the combined operations, and three (3) years and over for the intranasal operations.

First Group. Twenty (20) Cases Reported.—One had a double sac operation.

Of the twenty (20) cases two (2) were intranasal operations (Mosher), and eighteen (18) were the combined intranasal and external operations.

The two (2) intranasal operations were perfect results both as to pus and epiphora. Two (2) more intranasal cases out of a series of seven (7) have been traced. These also are perfect results. These cases are all three or more years operated.

There was one (1) failure in the eighteen (18) cases of the combined operation. This patient had tuberculosis of the sac (two cases of tuberculosis of the sac have been found in seventy-one [71] operations on the sac). She had temporary relief for a few weeks, but today, a year and a half after the operation, there is constant pus and epiphora.

There were two (2) partial failures in the eighteen (18) cases. The first of the two (2) was an old syphilitic. She was cured of the pus but still has some epiphora. The second patient had smallpox as a child and since that time has had a chronic blepharitis with narrowing of the palpebral fissure. This patient was operated on for epiphora only.

Both of these cases are to be put on probing.

Fifteen (15) of the eighteen (18) cases had perfect results in the cure of the pus and epiphora.

Three (3) of the eighteen (18) cases had a marked bow-string deformity of the scar.

Six (6) of the eighteen (18) cases had a slightly elevated or bowstring scar in some part of its course.

In all of the patients with deformed scars the incision was placed 6 mm. or less from the inner canthus and was made on a curve throughout. All of these were early cases.

Second Group. Twenty-two (22) Patients Reported. Twenty-four (24) sacs, two (2) patients had a double sac operation.

One (1) failure, a boy of nine, who had constant epiphora and no pus. This boy is to be given ether and explored with probe.

All the other cases were operated for pus and epiphora.

Twenty-two (22), or all the sacs which had pus, were cured of pus.

Seven (7), or about a third, had the epiphora markedly bettered but not cured. These cases are now in line for probing.

Nineteen (19) out of twenty-four (24) patients had no deformity of the scar.

Five (5) cases had a partial bowstring of the scar.

One (1) case had a marked bowstring deformity of the scar.

Three (3) of the partial failures in clearing up the epiphora had a mucous operation done subsequent to the sac operation. One case was bettered for one month, one (1) for three months, and one case has not been heard from.

COMBINED SUMMARY.

Four (4) out of seven (7) cases of intranasal operations on the tear sac are known to be a success, both as to the cure of pus and epiphora, after a period of three years.

Of forty-three (43) sacs reporting out of seventy (70) operated on, thirty-seven (37) were perfect results.

Two (2) were failures, one as to pus and epiphora, one as to epiphora only.

Nine (9) were partial successes as to epiphora.

Four (4) had marked bowstring deformity of the whole scar.

Eleven (11) had bowstring deformity of some part of the scar.

In other words, the pus was cured in over ninety per cent of the cases.

The epiphora was cured in 75 per cent.

Some deformity of the scar occurred in about a third of the cases. The deformed scars, however, were in the early cases in which the incision was made too near the inner canthus. The incision is now made at least 12 mm. from the inner canthus and it is made nearly straight. A flat, practically invisible scar is now the rule.

THE CAUSES OF FAILURE.

Five (5) of the nine (9) partial failures of the combined operation have been reexamined.

In one (1) case, a No. 5 probe passed easily into the nose. Patient has tearing once every hour.

In one (1) case there was a stricture of the lower canaliculus. Patient was tearing three or four times a day.

In one (1) case the lower punctum was tight and the probe would not pass the common opening of the canaliculi into the nose. Patient has epiphora every two hours.

In one (1) case water would syringe through the canaliculus into the nose, but a No. 2 probe would not pass. Patient has tearing once a week.

In one (1) case the probe passed but met with a narrowing at the common opening of the canaliculi into the nose. Patient has tearing three or four times a day.

It would seem, therefore, that the use of the probe or possibly the silver wire will be necessary in the after treatment of a certain number of cases operated by the combined method.

The post operative examination of these cases has shown that after the external operation—and the same should be true after the intranasal—it is not always easy for the probe to find the common opening of the canaliculi. The removal of the bony bed of the sac leaves its outer wall somewhat afloat. Therefore, in passing the probe, the canaliculus may kink ahead of it, and on looking into the nose the bulging wall of the sac is seen instead of the presenting end of the probe.

In the cases in which the combined operation has given only a partial success, Dr. Finck, who has been my mainstay in the follow up examination of the cases, has not felt like forcing the probe into the nose at the first examination if it passed hard. He feared making a false passage and endangering

whatever result had already been gained. Given a case in which the combined operation proved a total failure due to a stricture of the canaliculus or to a closure of the common opening of the canaliculi into the nose—the two chief causes of the partial failures—I should not hesitate to force the probe into the nose even at the first sitting. This would be the way in which I should first attempt to retrieve such a case. The probe once in the nose, I should follow it by the insertion of a silver wire style and leave this in place for some six weeks. The use of the style was a routine in my last intranasal cases.

The stricture of the canaliculus found in one of the cases antedated the operation and might have been caused by probing.

In the other cases the narrowing of the common opening of the canaliculi into the nose or its closure can be accounted for in two ways, trauma of the sac at the operation or narrowing afterwards as a result of the long standing infection.

A stricture of the canaliculus should be ruled out before operation. If the condition of the common opening of the canaliculi is not known, the degree of patency can be determined at the operation by syringing through the canaliculus after the inner wall of the sac has been removed. Probing should be tried at this time very gently for fear of making the familiar false passage.

Since the 42 cases reported nine more patients have been checked up. Two of these were private patients of mine. The rest were partly private and partly hospital cases of other operators. They change the statistics only for the better. One of my two in this supplementary list had a slight deformity of the scar. All the other cases had no deformity. One case which was operated for epiphora only showed no improvement for two months after the operation. Then the case was probed and the epiphora ceased. All the cases were cured of pus. One case which was operated for frequently recurring abscesses—this case is mentioned above—had laking of the inner canthus and but little epiphora. She has a permanently slit canaliculus and the epiphora and laking are unchanged.

There have been 71 cases of the combined operation in this vicinity. Forty-two of the cases are mine. Dr. C. T. Porter has done 15.

Of the 51 cases which have been checked up and are here reported, I operated 26, Dr. C. T. Porter operated 12, house officers under supervision 5, Dr. W. I. Wiggin 4, Dr. H. P. Finck 4, Dr. H. P. Cahill 1.

In a former reprint I have stated my indebtedness to Dr. Porter and to Dr. Finck for valuable suggestions. I wish again to express my thanks for their help.

CONCLUSIONS.

The follow up on these cases shows that both the intranasal and the combined intranasal and external operation will succeed and that their results to date—a period of two to three years—are permanent. The only drawback which the combined operation has developed is the deformity of the scar in the first cases. The later cases show that this can be avoided by properly placing the incision.

Having tried both operations—I first did a series of seven intranasal operations before I changed to the combined operation—I should compare the two operations as follows: Both will succeed; that is, both will cure the pus and the epiphora. The results of both operations are permanent. For me, at least, the intranasal operation is more difficult, is indirect, and to a great extent blind. There is always danger of opening the orbit by a misstroke and infecting it. The combined operation is easier than the intranasal. It is direct and done by sight. There is less danger of opening the orbit. If external drainage should become necessary for any reason, it is always possible to get it by reopening the incision. The intranasal operation, in my opinion, is not fitted to deal with the complicated cases (patients with permanent fistulæ or with partially removed sacs). The combined operation can deal successfully with every type of case.

One member of this audience is a strong rooter for the intranasal operation. This is natural because he was cured by it some six or seven years ago. I can easily understand his enthusiasm. It should be remembered, however, that all doctors prefer on themselves what they consider minor surgical measures. Another member of this audience is considering the combined operation. He is waiting for me to say the word when I feel that my results warrant it. I am ready for him if he is still of the same mind.

To conclude: In deciding which operation your patient shall have, do not think only in terms of rhinology or only in terms of ophthalmology. Be bigger than the "learned narrowness" of the single specialty; think in terms of both. Learn how to do the combined operation. If your instincts are surgical you will prefer it.

828 BEACON STREET.

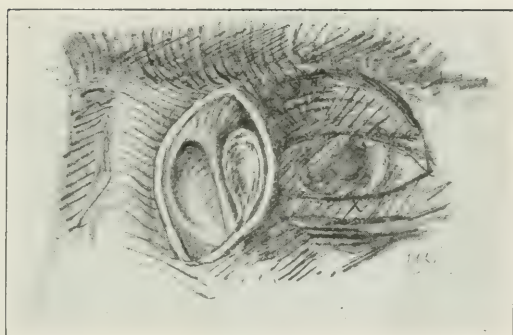


Fig. 1.
Drawing to show the sac turned out of its bed.

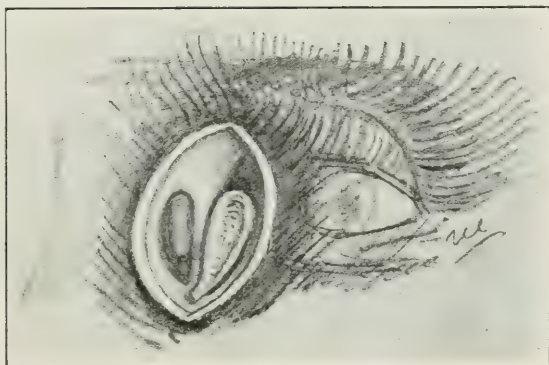


Fig. 2.

Drawing to show the opening made into the nose. In this opening the middle turbinate is seen. The anterior end of the turbinate has been removed.

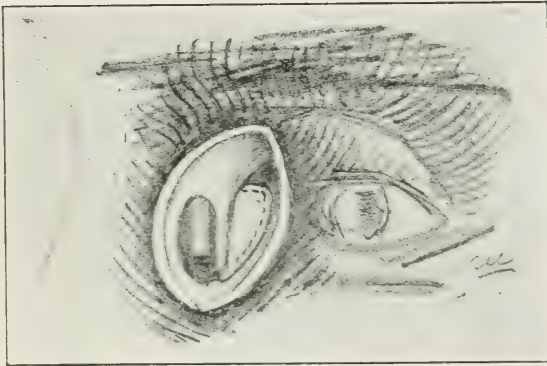


Fig. 3.

Drawing to show the incisions by which the sac is opened and its inner wall turned backward as a single flap. The dotted lines indicate the incisions.

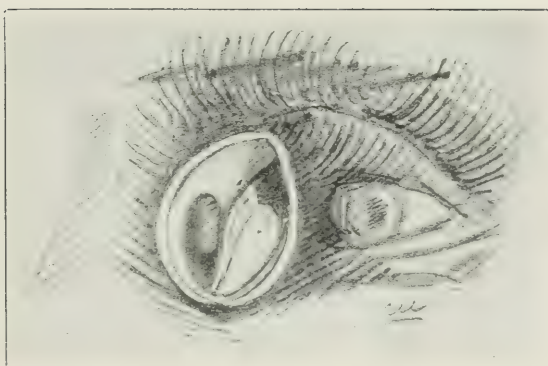


Fig. 4.

Drawing to show the inner wall of the sac turned upward and backward as a single flap. Making this flap is the first step in the removal of the inner wall.

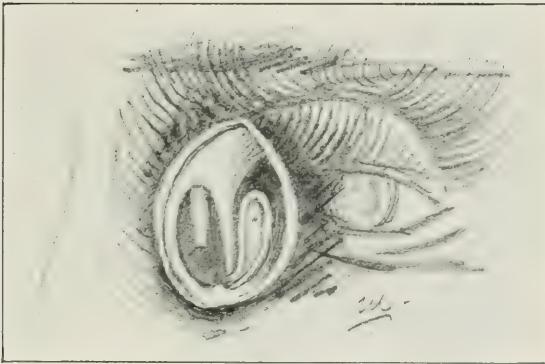


Fig. 5.

Drawing to show the completed operation. The inner wall of the sac has been removed. The inner wall of the bony canal in which the nasal duct lies has been removed down to the top of the inferior turbinate. The inner wall of the nasal duct has been removed.

DACRYORHINO CYSTOTOMY; COMBINED
METHODS.

BY W. E. SAUER, M. D.,

ST. LOUIS.

Our excuse for presenting this method is its simplicity, easy execution and satisfactory results.

There is probably a little misunderstanding about the terms "combined" and "intranasal" operations, as Mosher has just termed his The Combined Operation.

The operation I am about to describe is really an intranasal, with the probe passed into the sac from without. Since the time when West first introduced the intranasal operation, there have been a great many modifications. Our operation is, I believe, one of the simplest. Some operators, like Clark, introduce a probe after first making a stab incision into the sac from the skin, then doing an intranasal operation. The same principle is carried out by Blakesley of Kansas City, who makes a small incision and then introduces a trephine to make his entrance into the nose. So far as I know, he pays no attention to the interior of the nose, doing it simply by the sense of touch.

The operation which Dr. Wiener and I have been doing is the outcome of the rapid dilatation operation of Ziegler. The conception of our plan arose from passing a Ziegler probe in a case of congenital dacryocystitis in a girl, aged 4 years, April, 1911, in whom we found, on examining the nose, that the probe had not gone down the duct into the valve of Hasner, but had made a false passage through the lower end of the sac into the nose. It occurred to us then that it would be good practice to break through the nose intentionally in this manner and, utilizing the projecting end of the probe as a guide, which furnished a definite point on the inner wall of the sac, proceed to enlarge the opening upward, exposing the entire

nasal wall of the sac and affording even better drainage than that obtained through the small opening made by the Ziegler probe.

As shown in Plate 1, a Ziegler probe is first introduced through the inferior canaliculus into the lacrimal sac. The probe is then directed downward until it reaches the bottom of the sac, when it is turned inward at an angle of 45 degrees and then forced through the external nasal wall into the nasal cavity, as shown in Plate 2. In some cases a punctum dilator must be used before the Ziegler probe can be introduced. In a few cases we were unable to introduce the probe through the lower canaliculus, but had no difficulty in going through the upper one. In none of the cases did we find it necessary to slit the punctum.

The probe usually enters the nasal cavity just anterior to the anterior end of the middle turbinate, as in Plate 3. When the middle turbinate is large, it may be necessary to remove a portion of the anterior end immediately before the introduction of the probe. If the nose is narrowed by a septal deviation, a submucous resection must precede this operation, though in the majority of cases, while this is desirable, it is not necessary.

One of the great advantages of the Ziegler probe is that it shows the relative position of the sac. In all the nasal operations in which the Ziegler probe is not introduced there is no way of determining just where the sac is located. In some cases it is very prominent; in others it is impossible to tell the relation of the sac with the anterior end of the middle turbinate, for it is not constant. In some the anterior end covers the sac; in others it is directly in front of the middle turbinate; and in still others it is behind. Therefore, the position of the probe gives the exact position of the sac.

The next step is to withdraw the probe partially, as in Plate 3, leaving the point visible in the nose to serve as a guide for the removal of the bony covering of the canal and the lacrimal sac as far as necessary to establish a permanent opening. The opening made by the probe is then enlarged by means of an

electrically driven burr, about 2 mm. in diameter, as in Plate 4. The opening is enlarged upward and forward until it attains a diameter of between 8 to 10 mm. and is extended upward so that the probe may be introduced into the nose almost at right angles. That is, the handle of the probe, instead of being almost vertical as when pushed into the nose, may now be introduced in a horizontal position. It will now be found that a portion of the sac may be pushed through the bony opening into the nasal cavity by means of a probe, which is substituted for the Ziegler. (This is shown in Plates 5 and 6.) With the sac held in this position, biting forceps are introduced into the nose and as much of the sac as possible is grasped between the jaws and held there. The probe is removed and the jaws of the forceps closed, as shown in Plate 7. A portion of the sac is thus removed. The opening in the sac as well as that of the bone may be further enlarged by means of the forceps shown in Plate No. 8.

When the operation is completed the point of an Anel syringe is introduced in the canaliculus and the sac cavity thoroughly irrigated through the nose. This is repeated daily. The bleeding is usually very slight; in no case has packing been required. On the third day a probe is introduced into the nose. This may be required several times. Granulations are kept down by means of a 40 per cent solution of silver nitrate until the wound in the nose is entirely healed. The introduction of the probe and the applications of the silver solution are the chief points in the success of the operation. As in other similar operations, if the patient is not carefully watched, the opening may close.

With regard to the results, Mosher has just told you that results can be obtained with both the combined and intranasal methods. In 1914 West reported over 400 cases with 90 per cent successes with this method. If we can obtain such a result as that, the intranasal operation is certainly worth while. Our results have been fully as good as this with a much simpler method. The contraindications for this method, as Mosher also pointed out, are those cases in which the sac has been practically obliterated. In those cases I am willing to

agree with Mosher that his operation will come into place, but I cannot see any real objection to doing the intranasal operation prior to doing Mosher's operation, which is very much more favorable. The forcing of the probe from the bottom of the sac into the nose converts the West operation from one of great difficulty into a very simple procedure, by serving as a positive guide to the nasal location of the sac.

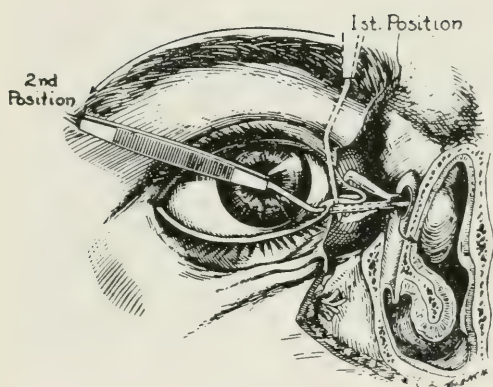


Fig. 1.
Path of probe from time of its introduction.

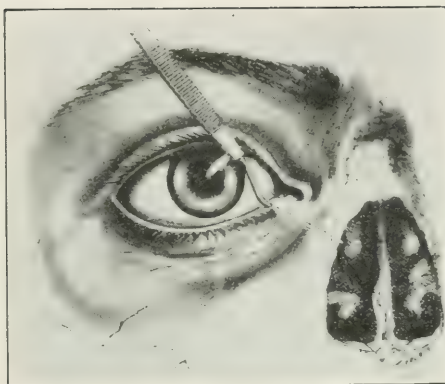


Fig. 2.

Direction of probe as it is forced into nose from bottom of sac.

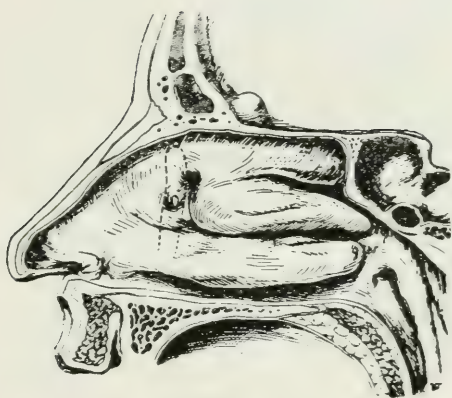


Fig. 3.

Position of probe as it emerges just anterior to middle turbinate. Dotted line is sac and duct.

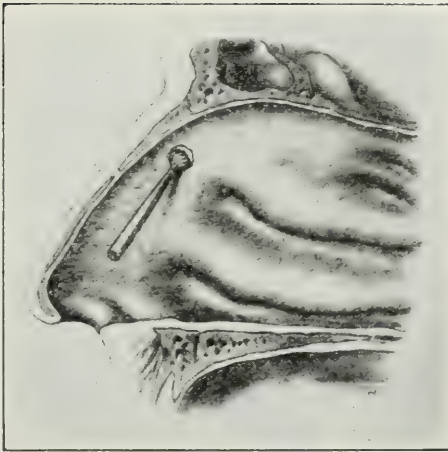


Fig. 4.

As probe is slowly withdrawn, the burr is introduced and the opening is sufficiently enlarged to give a good exposure of the sac.

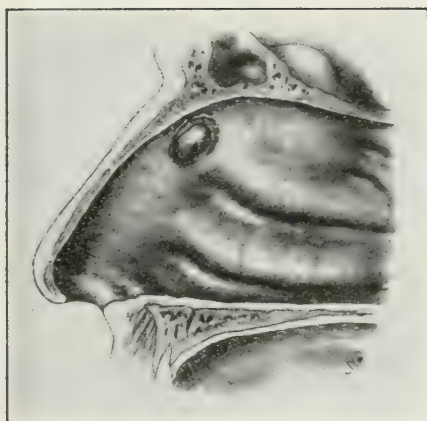


Fig. 5.
Showing the sac exposed.

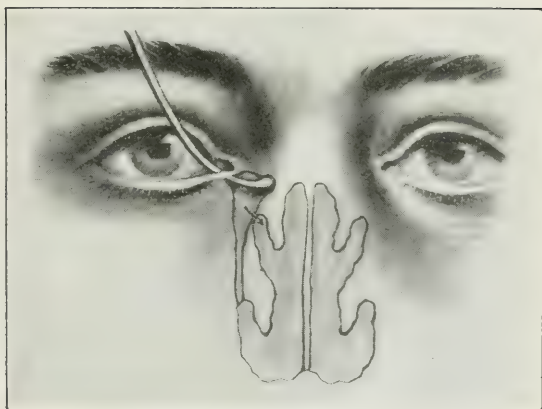


Fig. 6.

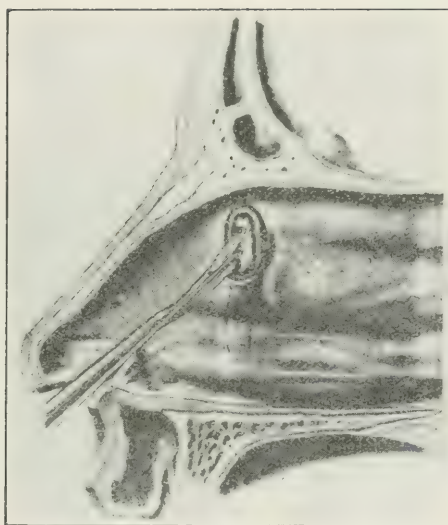


Fig. 7.

Sac is tented through opening with probe, and large piece punched out.

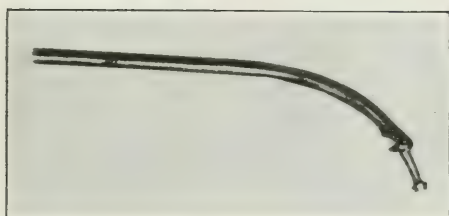


Fig. 8.

Punch forceps with which the opening of the sac may be further enlarged.

III.

STUDY OF REINFORCEMENT OF SOUND BY MEANS OF THE SCHAEFER RESONATORS.*

BY JOHN P. MINTON, PH. D., AND

ROBERT SONNENSCHN, M. D.,*

CHICAGO.

In previous papers by one of us (Sonnenschein), "Resonators, with Special Reference of the Schaefer Apparatus" and "Resonators as Possible Aid in Tuning Fork Tests—A Preliminary Report," the description and use of the Schaefer resonators were considered particularly from the viewpoint of the otologist. A consideration of the data given in these papers will show the value of this set of resonators to the otologist in cases of marked impairment of hearing. Many cases of deafness might easily be regarded as complete were it not for the establishment of the contrary through the increase in the loudness of the sounds from tuning forks by means of the resonators. Since their employment is to be strongly recommended, and since the Schaefer resonators have been used by one of the writers for a number of years in his professional work, it seems that the present paper on the sound intensity amplification of this set of resonators will be of much value in showing precisely the sound intensity over and above that of the forks alone.

By way of introduction, a few comments on resonators and resonance may be in place. Resonance makes it possible for periodic force to produce sympathetic vibrations. The periodic force may be blows of a hammer applied at the proper time intervals (what is referred to in physics as the proper phase relation) to a swinging pendulum; it may be the electric voltage or potential applied at the proper time intervals to an electric system, or it may be the acoustic energy applied at the proper time interval by a tuning fork to an air cavity of proper dimensions. In all these cases intense vibrations may,

under the proper conditions, be set up. The hammer blows may cause the pendulum to vibrate with large amplitudes, the electric force may cause a periodic flow of electric current that may become very intense when the electric constants of the circuit are properly adjusted, and in a similar manner the periodic force from a tuning fork may cause intense sounds to be produced at the resonator. So that in all the branches of physics the phenomenon of resonance and the question of resonators are of much importance, and physicists are called upon constantly to deal with them.

To be specific, let us suppose that in the case of sound the periodic force is produced by the vibrations of a tuning fork. This causes sound waves to be generated in the surrounding air, and they cause the second body, called the resonator, to vibrate with the same period as the tuning fork. Thus, the second body may be a tuning fork, a column of air enclosed in a pipe, etc. Under the proper conditions comparatively intense vibrations may be set up in the second body and sounds of particular pitches with greatly increased loudness are produced. In fact, in acoustics this is the chief object of resonators.

As an example of this action of a resonator, we may take the case of the increase in the loudness of the tone given by a tuning fork when it is placed in front of one of the Schaefer resonators which has been adjusted to the pitch of the fork. The fact that the prongs of the fork have not a very great area explains why they are not capable of setting into intense vibration a large amount of the surrounding air because of the slippage of air back and forth around the prongs as they vibrate. In addition, the two prongs of the fork are moving in opposite directions, so that there is interference between the waves emitted by the two prongs. This interference causes a still further reduction in the intensity of the vibrations in the surrounding air. If, however, the tuning fork is held in front of the resonator, then the column of air within will be set into marked vibration, due to the phenomenon of resonance, and an intense radiation of sound energy will occur; provided, of course, that the open end of the resonator is not too small, for in that case the external air would receive a smaller portion of the acoustic energy within the resonator.

In passing, one additional example of resonance may be cited, namely, that of a string: when the latter is vibrating freely there is a very small amount of air set into vibration because of the small superficial area of the string. In addition, the waves thus produced interfere with one another because the condensation on one side of the string interferes with the rarefaction on the opposite side. However, if the string is connected to the walls of a resonator so that the vibrations are communicated directly by the walls to the enclosed air column intensified vibrations are set up in the surrounding air.

These are examples of the intensifying of sound by true resonance. To demonstrate this fact, it would only be necessary to substitute a resonator which responds to a different tone and then no reinforcement of the sound would be observed. These cases should not be confounded with those of forced vibrations with a corresponding increase in loudness. Thus, a table top will cause an intensification of sound given off by all the forks of lower pitch, and tones which might otherwise be inaudible are capable of being heard by a large audience. The intensification of the sound is due, of course, to the surface of the table setting into violent vibration a large volume of surrounding air.

It is important to remember that the resonator receives the energy necessary to set it into violent vibration from the sounding body acting as the periodic force—for instance, that of the tuning fork. Therefore, since the increased loudness of the emitted sound is due to the resonator means that more sound energy is radiated per unit time into the air, it follows that the sounding body (the tuning fork) must lose its energy of vibration more rapidly when a resonator is present than it does without the resonator. That this is so can be easily demonstrated: for example, a 512 d. v. fork was heard 30 seconds when placed close to the ear and was heard only 20 seconds when the resonator was used. Again, a 256 d. v. fork was heard 120 seconds without the resonator and only about 90 to 100 seconds with the resonator. Lord Rayleigh cites a more convincing case observed by Koenig: "Thus, without a resonator a fork of 256 double vibrations sounded in a satisfactory manner for about 90 seconds. A resonator of adjust-

able pitch was then brought into proximity, and the pitch, originally much graver than that of the fork, was gradually raised. Even when the resonator was still a minor third below the fork there was a slight diminution observed in the duration of the vibratory movement. As the natural tone of the resonator approached nearer to that of the fork, this diminution in the time became more pronounced up to the immediate neighborhood of unison; but at the moment when unison was established the sound was powerfully reinforced; but this exaggerated intensity fell off rapidly and the vibration died away after eight to ten seconds. The pitch of the resonator being raised, the duration of the vibrations gradually recovered its original value of about 90 seconds."

These data are important to remember in the use of resonators, for if the tones are louder one might expect that they would be heard a correspondingly longer time. The above data show this not to be the facts, but on the contrary they should be heard by a normal ear a shorter time interval with the resonator than without it. In the case of persons with impaired hearing consistent results should be observed with any given patient at one pitch. Results would be inconsistent when considered as a whole, for some patients would hear longer with the resonator than without it, while the reverse should be expected for patients with a different impairment of hearing. The findings at various pitches for any one patient need not be consistent, inasmuch as the impairment is not the same at different pitches. We believe this will explain satisfactorily some of the data (which may appear inconsistent) observed and reported by one of the present writers. Hence, if it is desired to make any observations of the duration of hearing with the resonators consistent results will be obtained when a comparison is made only with the time heard by normal ears with the resonators. No effort should be made to obtain a comparison between the times heard with and without the resonator.

Experimental Results of Intensity Amplification.—The method of employing the Schaefer resonators, a photograph of which is shown, has been described in the papers to which reference has already been made. This method consists in attaching a rubber tube to the closed end of the resonator and

then connecting with the patient's ear by means of a hard rubber olive tip. The vibrating fork is held by the examiner before the open end of the resonator and the sound is reinforced. The amount of accentuation, however, will be different for the patient than for the examiner because both persons are not listening to the sound in the same manner. In order that the examiner may not draw doubtful conclusions because of this fact, the following three groups of tests were made:

GROUP I. AMPLIFICATION AS PERCEIVED BY THE EXAMINER.

The forks were excited by a rubber stopper having a brass core about 2 cm. long and 1 cm. in diameter. To obtain a uniform force the stopper was attached to a string 48.5 cm. long and the opposite end suspended from a suitable arm. The cork was pulled aside certain definite distances and allowed to swing freely through a measured arc. The forks were held at the bottom of the arc so that the force of the impacts could be calculated.

Tests for the purpose of determining the amplification of the sound as heard by the examiner were made in the following manner: A current from an audion oscillator was passed through a telephone receiver, the frequency of the current having first been brought into unison with that of the fork. A galvanometer was used to measure the current necessary for the receiver to generate a tone of the same loudness as that given forth by the resonator when the fork was held before the open end (a in Table I).

In this instance the receiver diaphragm and the open end of the resonator are the same distance from the examiner's ear (usually 60 cm.). In the second part of the test (b in table I) the receiver and fork were placed at the same distance (usually 12.5 cm.) from the ear and the current noted that caused the tone from the receiver and the fork to sound equally loud. Care was taken to strike the forks with the same force in both parts of the test. In passing, it should be noted that the forks of different pitches in some instances had to be struck with different forces in order to obtain the desired comparison. The particular forces necessary for this purpose were determined by trial and depended on both the receiver characteristics and the dimensions of the forks, as

shown in Table V. The sensitivity of the galvanometer also entered into the selection of the forces.

The square of the ratio of the current through the receiver when the resonator was used to that when the resonator was not employed gave a direct measure of the relative intensity of the tone of the tuning fork when used with and without the resonator. In other words, the square of this current ratio will show the intensity reinforcement of the sound by the resonator at the moment when the fork was placed before the opening. This, of course, is the maximum reinforcement.

It may be said, parenthetically, that the square of the current ratio is sought because the sound intensity produced by the receiver diaphragm is proportional to the square of the electric current.

TABLE I.
RESONATOR TESTS.

Test No. I.—Amplification of sound as it appears to the examiner.

Ratio of current with closed resonator (not connected with ear) and that with fork at constant distance from examiner's ear.

(a) Test with resonator, fork and telephone receiver.

(b) Test with fork and receiver only.

N. B.—Type Western Electric 144 telephone receiver used in all tests.

Fork used		Distance of receiver and resonator from ear	Distance of swing of rubber weight against fork	Velocity at time of impact against fork	Length of resonator	Intensity amplification as it appears to examiner
d sharp	(a)	25 Cm.	4.5 Cm.	20 Cm. per	553 mm.	15.5
154 v. d.	(b)	7 Cm.	4.5 Cm.	sec.		
c1	(a)	60 Cm.	6 Cm.	27 Cm. per	327 mm.	96.5
256 d. v.	(b)	12.5 Cm.	6 Cm.	sec.		
g1	(a)	60 Cm.	9 Cm.	40 Cm. per	213 mm.	222
384 d. v.	(b)	12.5 Cm.	9 Cm.	sec.		
(small fork)	(a)	60 Cm.	45 Cm.	185 Cm. per	185 mm.	273
435 d. v.	(b)	12.5 Cm.	45 Cm.	sec.		
c2	(a)	60 Cm.	20 Cm.	90 Cm. per	145 mm.	204
512 d. v.	(b)	12.5 Cm.	20 Cm.	sec.		
g2	(a)	60 Cm.	45 Cm.	185 Cm. per	96.5 mm.	62
768 d. v.	(b)	12.5 Cm.	45 Cm.	sec.		
c3	(a)	60 Cm.	45 Cm.	185 Cm. per	64 mm.	68
1024 d. v.	(b)	12.5 Cm.	45 Cm.	sec.		

Table I shows the results that were obtained for the four resonators in this group of tests. It will be observed that the reinforcement of the sound varies from 15.5 to 68 and that the efficiency of the resonators diminished rapidly for both the higher and lower pitched resonators. The maximum intensification is obtained at the medium pitches, reaching a value of 273 for the a^1 (435 d. v.) fork.

GROUP II. AMPLIFICATION AS PERCEIVED BY THE PATIENT.

For this series of tests the forks were excited as above described and held before the open end of the resonator while a small rubber tube about 75 cm. long attached to the closed end of the resonator was connected with the ear by means of an olive tip. A tone of the same pitch as that of the fork was given forth by the receiver placed in contact with the ear. The loudness of this tone was made equal to that of the resonator and the corresponding current noted. In the second part of this test the fork was excited with the same force as before and then held in close proximity to the observer's ear. The loudness of the tone was carefully noted, and immediately afterwards the telephone receiver, with a current of the proper frequency passing through it, was placed against the ear. Repeated comparisons were made, and when equality of the two tones was reached the receiver current was again measured. The square of the current ratio in the two instances gave the intensity amplification produced by the resonator when used in this manner.

Table II shows the same results that were obtained with the four resonators. It will be observed that the amplification increases from 2.9 for the highest pitch to 44 for the g^1 (364 d. v.) and then decreases to 10.3 for the d sharp (154 d. v.) fork.

These data are in accord with the statements made by Schaefer in one of his papers in which, however, he gave no measurements to confirm his assertions. It may also be remarked that no data regarding the intensity amplification of sound by resonators have been found by us in either medical or physical references.

TABLE II.

RESONATOR TESTS.

Test No. II.—Amplification of sound as it appears to the patient.

Ratio of current with

(a) Receiver held against ear and resonator connected with ear via rubber tubing, and

(b) With receiver against ear and fork held close to ear.

N. B.—Type Western Electric 144 telephone receiver used in all tests.

Fork used		Distance of rubbing of weight against fork	Velocity at time of impact against fork	Length of resonator	Intensity amplification as it appears to patient
d sharp	(a)	10 Cm.	47 Cm. per sec.	553 mm.	10.3
154 d. v.	(b)	10 Cm.			
e1	(a)	6 Cm.	27 Cm. per sec.	327 mm.	38
256 d. v.	(b)	6 Cm.			
g1	(a)	9 Cm.	40 Cm. per sec.	213 mm.	44
384 d. v.	(b)	9 Cm.			
(small a1 fork)	(a)	45 Cm.	185 Cm. per sec.	185 mm.	25
435 d. v.	(b)	45 Cm.			
e2	(a)	20 Cm.	90 Cm. per sec.	145 mm.	10
512 d. v.	(b)	20 Cm.			
g2	(a)	45 Cm.	185 Cm. per sec.	96.5 mm.	9
768 d. v.	(b)	45 Cm.			
c3	(a)	45 Cm.	185 Cm. per sec.	64 mm.	2.90
1024 d. v.	(b)	45 Cm.			

GROUP III. INTENSITY AMPLIFICATION IN UNCONFINED OPEN SPACE.

The above tests were made in a room, and it was desired, as a further experiment, to determine the amplification ratio in an open, unconfined space. This third condition of use is quite different from the two just described, and for this reason it is to be expected that the findings would be greatly modified. In an unconfined space there is a direct radial propagation outward of the sound energy at distances large compared with the dimensions of the forks and resonators. Under this condition there are no reflections from neighboring

surfaces, and furthermore there are no standing waves to contend with. Under these conditions the sound intensity would vary inversely as the square of the distance from the source.

The procedure, then, was to obtain a direct comparison between the greatest distances a fork could be heard (a) without and (b) with the resonator. The square of the ratio of these distances gives a direct measure of the intensity amplification. Care was taken to excite the fork with the same force in the two instances. In (a) the fork was struck and the distance noted at which the tone was just audible. In (b) the fork was struck and held before the resonator and the distance again noted at which the tone was just audible. The square of the ratio of these two distances gave the desired result.

The findings under these conditions are recorded for the four resonators in Table III. This method of use also shows that the resonators are much more efficient for sound reinforcement at the lower pitches than at the higher ones, but that the decrease at the lowest pitches is very much less marked under the present condition than under the two preceding ones.

The variation of the efficiency of the resonator to reinforce sounds of different pitches is a phenomenon familiar to physicists. The main reason for the reinforcement, as has been pointed out earlier in the paper, is because of the proper phase relation of the pressure due to the fork with that due to the resonator. That is, the phase relation must be favorable in order that a reinforcement may occur. When the wave length of the tones becomes comparable with the dimensions of the fork and with those of the resonators, then the phase relations deviates from the proper ones, and thus large intensification by a resonator of the tones of the higher pitches becomes impossible of attainment.

The interference of the sound in the air surrounding the forks (already referred to in the paper) is more marked for the forks of the lower pitch than those of higher pitch. This fact is responsible to a large extent for the decrease in the intensity amplification of the resonators of a lower pitch. Hence, the phase relations at both lower and upper extremities of possible resonance due to sound resonators become the determining factors in limiting the intensity of the sound.

TABLE III.

RESONATOR TESTS.

Test No. III.—Amplification of sounds in unconfined space.

Forks struck with rubber hammer and distance noted at which they were heard

(a) without resonator

(b) when held near corresponding resonator.

Resonators were used at height of 70 cm. from the ground. (One control test at height of 600 cm. from ground showed no material difference in amplification.)

Fork used	Distance heard (a) without resonator	Distance heard (b) with resonator	Length of resonator	Intensity amplification
(1) A 108 d. v.	16.5 Cm.	1550 Cm.	786 mm.	8900
(3) d sharp 154 d. v.	30 Cm.	3100 Cm.	542.5 mm.	10700
(2) c 128 d. v.	19 Cm.	1450 Cm.	668 mm.	5800
(4) c1 256 d. v.	52.5 Cm.	4745 Cm.	330 mm.	8225
(5) e1 320 d. v.	136.5 Cm.	4405 Cm.	257.5 mm.	1047
(6) g1 384 d. v.	53.5 Cm.	2408 Cm.	213 mm.	1460
(7) (small a1 fork) 435 d. v.	10 Cm.	330 Cm.	185 mm.	1095
(8) e2 512 d. v.	140 Cm.	4067 Cm.	145 mm.	845
(9) g2 768 d. v.	1272 Cm.	5156 Cm.	96.5 mm.	16.6
(10) c3 1024 d. v.	2350 Cm.	5209 Cm.	64 mm.	5
e2 512 d. v. CONTROL TEST Resonator 600 Cm. above ground.	143 Cm.	4600 Cm.	145 mm.	1030

TABLE IV.

RESONATOR TESTS.

Comparison of intensity amplifications as they appear to the examiner, the patient and in open unconfined space.

Fork used	Amplification as it appears to examiner	Amplification as it appears to patient	Amplification in open unconfined space
d sharp	15.5	10.3	10700
154 d. v.			
c1	96.5	38	8225
256 d. v.			
g1	222	44	1460
384 d. v.			
(small			
a1 fork)	273	25	1095
435 d. v.			
c2	204	10	845
512 d. v.			
g2	62	9	16.6
768 d. v.			
c3	68	2.85	5
1024 d. v.			

A final table is given below, the purpose of which is to bring together for direct comparison the intensity amplification of the four resonators when tested according to the three methods outlined above. The reinforcement of all tones is greatest in the open, unconfined space, is consistently the least as observed by the patient and is intermediate as heard by the examiner under actual testing conditions. Since the intensity amplification is much greater when the resonators are used in the manner as tested in the unconfined space, it might be advantageous to make use of this fact in testing deafness. This could be done merely by comparing the distances that a fork, when sounded before a resonator of the proper pitch, could be heard by the normal and the defective ears. The ratio of these distances, when squared, will show the intensity amplification required for the deaf person to hear compared with that required by a normal ear.

Finally, it may be pointed out that a set of standing waves are present in the rubber tube leading to the ear and attached to the resonator. The distance between the nodes and loops within the tube will be determined by the pitch of the tone. These loops and nodes could be detected with the ear when the fork was sounded before the resonator and the tube compressed at different places. Compression at a node (zero displacement of the air) did not change the loudness of the tone.

but compression at a loop (maximum displacement of the air) caused a decided change in the intensity of the tone as observed by the ear in the vicinity of the resonator.

TABLE V.

DIMENSIONS OF PRONGS OF FORKS USED.

		Width	Length	Thickness
A		1.80 Cm.	23.75 Cm.	0.8 Cm.
108 d. v.				
c		1.9 Cm.	30.3 Cm.	1.5 Cm.
128 d. v.				
e1		1.45 Cm.	16.15 Cm.	0.8 Cm.
256 d. v.				
e1		1.4 Cm.	12.6 Cm.	0.6 Cm.
320 d. v.				
g1		1.5 Cm.	11.7 Cm.	0.65 Cm.
384 d. v.				
a1		0.9 Cm.	7.7 Cm.	0.3 Cm.
435 d. v.				
Weighted:	Size of weights	1.7 Cm. wide		
c2	Fixed weight	2.8 Cm. long		
512 d. v.		1.8 Cm. thick		
g2		1.45 Cm.	8.1 Cm.	0.65 Cm.
768 d. v.				
c3		1.45 Cm.	7.0 Cm.	0.65 Cm.
1024 d. v.				
c1	Weights	1.65 Cm. wide		
weighted	(adjustable)	1.45 Cm. long		
Dis. 154 d. v.		2.55 Cm. thick		
		1.55 Cm.	14.5 Cm.	0.65 Cm.

Now, when the olive tip is placed in the patient's ear it probably acts to some extent as though the end of the tube were closed (but not entirely, for the meatus is much larger than the hole in the olive tip). This would tend to make the end of the tube act as a node of displacement (or a loop of pressure), whereas if the tube were open the end would act like a loop of displacement (or a node of pressure). For these reasons at 512 d. v. we made a measurement of the amplification ratio first with the olive tip placed at a loop and next at a node of displacement. These positions were determined with the olive tip not inserted in the meatus. In the first case the intensity amplification was 10 and in the second case 11.5 as observed by the method of test used on the patients. This difference of about 10 per cent is within the limit of error of the observation reported herein, so that we may conclude with much certainty that the length of the rubber tube is, physically speaking, not a factor of much importance. It is advised, however, that an excessive length of tube be avoided, inasmuch as it changes the pitch of the resonator to a small degree. For this reason the exact setting of the resonator for

any particular fork should be determined under actual testing conditions.

CONCLUSIONS.

1. The sound intensity amplification of the (Schaefer) resonators depends on the manner in which they are used. The maximum value of this amplification as observed, firstly, by the examiner, is 273; secondly, by the patient, 44, and thirdly, as it appears in unconfined space, 10,700.

2. The efficiency of the Schaefer apparatus as a resonator decreases rapidly for the higher pitched tones. The same is true at the lower pitched ones except when tested in the open, unconfined space.

3. The resonators do not increase the total energy given forth by the tuning fork, but permits this energy to be dissipated at a greater rate.

4. The decrease of the efficiency of the resonator with rising pitch is due to the unfavorable phase relation of the air pressure produced by the tuning fork with that caused by the resonator.

5. Inasmuch as the resonators cause tones to be produced of greatly reinforced intensity, they might be of considerable value from a clinical viewpoint. This is particularly true for the set of four Schaefer resonators, since there are given herein the actual magnitudes of the intensity amplification. As noted in previous papers, the Schaefer resonators lend themselves well to practical purposes, since they cover a comprehensive range, are comparatively cheap, compact and easily manipulated.

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IV.

REGIONAL ANATOMY OF THE TEAR SAC.

BY JAS. M. PATTON, M. D., F. A. C. S.,

OMAHA.

Any consideration of the regional anatomy of the tear sac must recognize the importance of the bony lacrimal fossa and its continuation, the nasolacrimal canal. An examination of a considerable number of dried skulls shows rather marked variations of structure. For example, the anterior and posterior crests may be comparatively flat and the fossa correspondingly shallow, or they may be so pronounced as to make the fossa very deep, and in some instances may even give it the appearance of an incomplete cylinder. This, of course, is of little significance except that where we find such abnormal conditions in the fossa we are liable to find abnormalities in the bony lacrimal canal. For instance, the opening from the fossa may be much smaller than normal, or it may be irregular in shape and position. The makeup of the floor of the fossa is of greater interest from a practical standpoint. If the lacrimal bone predominates, as shown in Figure 1, the floor is fragile, easily broken down, and the fossa is more likely to be at least partly covered on the nasal side by the anterior tip of the middle turbinate. Where the ascending process of the maxilla predominates, as shown in Figure 2, the floor of the fossa is very dense and in many cases marked difficulty will be experienced either in passing from the sac through into the nose, as in the Wiener operation, or in coming from the nose through into the sac, as practiced by Yankauer and others. In an examination of about one hundred and fifty orbits, we found that sixty-eight were of the porous type. In the remaining forty-seven the maxillary process predominated. I was unable to judge from the contour of the bony skull in every case as to just what the condition of the fossa would be, but it was quite evident that where there was a marked development of the heavy bones of the face, jaws, fron-

tal prominences, etc., that the maxillary element of the fossa was more likely to predominate, while in the lighter skulls the lacrimal element was more in evidence.

In operating on these cases we should remember that the lacrimal sac does not come directly in contact either with the bony fossa or with the overlying muscles, but is partially inclosed by an extension of the periorbita, which on the nasal side lines the fossa and at the posterior lacrimal crests divides to form the lacrimal fascia, which is rather firmly stretched, roofing the fossa between the two crests, and may be mistaken for the sac itself by inexperienced operators. There is a narrow space between the sac and this fibrous envelope occupied by vessels and lymphatics. According to Whitman, the canaliculæ never join till after passing the fascia lacrimals and the so-called common canaliculus is in reality a diverticulum from the sac.

It is the opinion of our anatomical confreres that the flattening of the upper extremity of the sac is due to compression by the fascia lacrimals, aided to a certain extent by the action of the parslacrimalus muscle (Horners) and the medial palpebral ligament, the latter forming an excellent landmark for the location of the upper third of the sac. In any case, instead of the lacrimal sac being somewhat larger than the diameter of the bony canal—which it undoubtedly is in some pathologic states—it may have more of the shape of an irregular cone with its base at the bony opening of the canal, as Shaffer has shown, from his reconstruction, there may be marked variations from the so-called normal with constrictions, angulations and diverticula of both the bony and membranous tract, undoubtedly accounting for some of our failures in attempting to restore drainage through regular channels.

Figure 3 from Whitman is of interest and shows that the axis of the canal is inclined backwards approximately fifteen to twenty-five degrees from the vertical axis of the face. The direction from the inner canthus is toward the space between the anterior border of the first and third molar teeth. It also shows in the insert the relation of the canals to the antra, with the so-called lacrimal protuberances.

The nasal opening of the canal varies considerably as to size and location. The "protecting valves" are simply folds of the

lining mucosa and without any direct valve action. According to Whitman, the mucosa is continued from the nose to the palpebral opening of the canaliculæ and convinces a number of careful observers that the large majority of lacrimal infections are of nasal origin.

In preparing for a dacryorhinocystotomy we may well consider the probable character of the bony lacrimal fossa, whether it is fragile or dense, the *facia lacrimals* and its overlying medial palpebral ligament, the probable size and direction of the nasal lacrimal duct with its point of emergence beneath the inferior turbinate and the probable presence or absence of overlying anterior ethmoidal cells. The recognition of these anatomical conditions may help us to decide as to which of a number of excellent operative procedures is best suited for the correction of the symptoms we are attempting to relieve.



Fig. 1.

Left orbit showing lacrimal bone occupying nearly all of lacrimal fossa. Bone porous. Easily broken down.

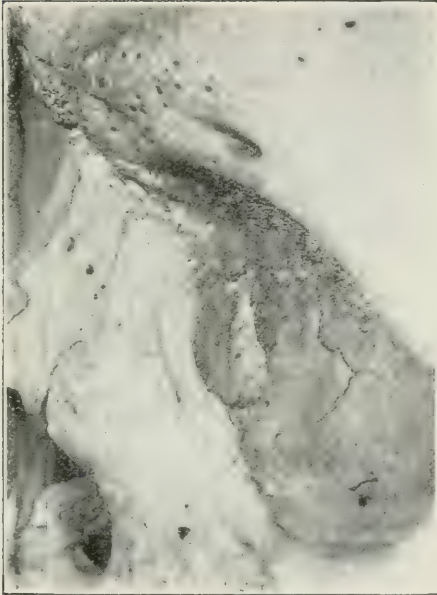


Fig. 2.

Left orbit showing floor of fossa almost entirely made up of ascending process of maxilla. Floor very dense and thick. Note heavy supraorbital margin.



Fig. 3.

From Whitman, showing angle of nasal lacrymal duct and in insert cross section with relation of nasal lacrymal ducts to antra.

V.

INDICATIONS, CONTRAINDICATIONS AND PREPARATION FOR DACRYOCYSTORHINOSTOMY.

BY DR. RALPH A. FENTON,

PORTLAND, OREGON.

The operation of dacryocystorhinostomy is only to be considered in cases where simpler means of treatment of tear sac suppuration have not sufficed. Indications for it must be based upon very careful anatomic study and, as the previous paper has demonstrated, the anatomy may be very intricate. The nose should always be examined both before and after careful shrinkage by adrenalin, and those who do only eye work or nose work should hesitate to select this procedure without consultation by a colleague prepared in the other branch.

A few diagrams, trespassing a little upon the field which Dr. Patton has covered with such admirable detail, will perhaps clarify the matter of indications and contraindications.

As opposed to excision or obliteration, the first indication (Fig. 1) is for speedy, radical cure. Dangers to be avoided include those arising from a pus focus that cannot be kept clean; septic absorption from the sac itself, a closed cavity often without drainage for weeks at a time; consequent iritis, endocarditis, arthritis and the like. Locally, there is constant danger of *ulcus serpens* or other corneal involvement; and, due to drainage into the nose, we may have destruction of normal epithelium, with septal ulcerations and even *ozena*; or, still further down, we may find lateral pharyngitis, or recurrent tonsillitis and laryngitis. Indeed, an ethmoiditis or maxillary antrum involvement is not infrequently associated with chronic dacryocystitis.

The second indication (Fig. 2) is based on the need for a clean eye. No intraocular operation of any moment may be done in the presence of severe suppuration. The pus may be temporarily shut off by ligation of the canaliculi, an uncertain

makeshift when more modern means such as dacryocystorhinostomy are available. Chronic conjunctivitis, blepharitis, fissures and dermatitis are one or all present, and at all times the hazard of recurrent phlegmon and its extension into erysipelas must be reckoned with—an especial danger in those with thin and florid skin.

Third (Fig. 3) comes the need for restoration of physiologic function. The proposition that gradual lessening in the amount of tears secreted will follow excision of the sac is probably based upon the observation made by many operators that cessation of chronic conjunctival irritation has taken place at the same time. In the presence of chronic conjunctivitis a constant stimulus to lacrimation naturally remains, and may even suggest the removal of the lacrimal gland if all other measures have failed. Further interferences with physiologic function come by reason of diverticula of the sac, often extending far out into the cheek. Cicatricial stenoses may have arisen from injudicious treatment, overstretching, or from altogether false passages, particularly in those cases mentioned by Schaefer where duct and sac join at right angles. Anywhere along the duct bony stenosis may occur, with new bone formation and complete occlusion. Obliteration or excision cannot, of course, restore, but must destroy the further physiologic use of these structures.

Coming now (Fig. 4) to the need for good cosmetic results: Successful dacryocystorhinostomy will avoid excessive tearing with its entailed skin irritations. People who are obliged to be out of doors a great deal become very much annoyed by the necessity for wiping the eye, by clouding of glasses, and by the dermatitis that frequently arises. Moreover, by selection of appropriate type of operation, the external scar may be avoided or kept practically invisible.

With reference to the type of operative technic selected, three essentials must be remembered:

- a. The nose must be roomy, or must be made so;
- b. There must be one functioning canaliculus;
- c. There must be enough of the sac left to form a good drainage channel.

Unless these things are assured, it is very probable that failure will attend any attempt to perform the various types of dacryocystorhinostomy.

Turning to the contraindications, we may first regard the occlusions of infancy, ordinarily disappearing unaided with the lapse of time or after a single probing. These things get well and do not call for extensive invasion of the rapidly growing bones of the face. Similar conservatism should attend the much more virulent disorders of old age, by reason of the brittleness of the bones, as well as danger to the lungs and heart from anesthesia. Here palliation has its proper field. It is of course possible, even with most careful instrumentation, so to fracture the orbitonasal wall that invasion of subcutaneous planes may occur, bringing on erysipelas or cellulitis vastly dangerous to those along in years.

Examining the ocular contraindications (Fig. 5), we find obliteration of the canaliculi, fibrous shrinkage of the sac and caries of the bony wall, resulting from improper probing as well as from syphilis. Existence of lupus or of cancer, even though slow growing, should in this region be deemed an absolute contraindication.

Cardiovascular, renal or diabetic conditions, and of course tuberculosis or syphilis, must guide the decision for persons requiring general anesthesia. Wise policy will suggest the use of palliative treatment while the cure of the general disorder is awaited.

Keloidal tendencies in the colored race must be considered before any operation is chosen that will entail going through the skin. Persons with thin, florid skin, readily chapped, must be watched for erysipelas. Others, with pyodermic tendencies—acne, boils, impetigo and the like—must lead us to consider well the danger in permitting such a surface infection to invade the loose fascial planes of the orbitonasal region.

Considering now the nasal contraindications (Fig. 6), which may in many cases be remedied by operation, we may first examine the overlying groups of ethmoid cells. In the diagrams many structures at different depths are drawn in side by side—unciform, agger nasi or bullar cells, and cystic degenerations of the middle turbinate. These structures must be carefully worked out, a task often facilitated by radiographs.

In doubtful cases bismuth is sometimes injected into the sac, but this is rarely needed. The large middle turbinate which interferes with access to the lacrimal bone, or to the crest of the ascending process of the superior maxilla, must be reduced in volume or resected in part—measures occasionally needed for the inferior turbinate as well.

High septal thickening which impinges against the middle turbinate often completely prohibits access to the lacrimal sac. Occlusive or even moderately narrowing anterior deviations, whether cartilaginous or involving the crest, should be done away with also. Thickening of the ascending process of the superior maxilla frequently protrudes very markedly into the nose, and it is extremely difficult, without removal of this thickening as a first step in the operation, to get anywhere in the vicinity of the sac in such a way that it may be inspected and cut off or even seen when pushed into the nasal opening.

The contraindication of pus in the nasal operative field is most important: pus flowing up and out from the maxillary antrum, down from the frontal sinus and forward from the anterior ethmoids, streaming across the middle turbinate and accumulating along the floor of the nose. Operation through such a field with any intranasal invasion of the lacrimal tract is surgically rather undesirable, in spite of high local resistance and the good drainage usually present. Nasal polypi, proceeding from the antrum, ethmoids or frontal, are not only obstructive to the field, but usually mask a flow of pus that needs clearing up. Papillomatous or "bleeding polypi" must entail the same careful reservations as to intranasal procedures which they invite when found near the eye, externally. Syphilitic or tuberculous intranasal disease also forbids the choice of dacryocystorhinostomy.

With respect to all preliminary nasal operations, it may be said that if such surgical preparation is easy and readily applicable, it should certainly be done at least two to four weeks prior to the time set for the tear sac operation. If this is not done, if the second operation comes too close to the first, there is very often more or less serious interference by crusts or granulations with proper drainage of the nasal aspect of the wound. Opened ethmoid cells, in particular, should be cut well back out of the field of the tear sac procedure.

As to the preparation, we should consider the general medical aspects of the patient, especially when advanced in years. Blood pressure should be reduced and albuminuria and acidosis forestalled by proper treatment and dietary regulation. We must also avoid the onset of dangerous renal complications while these people are in the hospital. Duration of the stay in hospital is sometimes very limited, and it is quite possible that an occasional serious complication reported might have been avoided by keeping such a patient quiet under hospital observation for a longer period. The time will naturally vary with the extent and nature of the operative procedure.

Hypodermic use of morphin, usually with atropin, customarily precedes general or local anesthesia for this group of operations.

Preparation of the eye varies of course with the type of operation and the choice of the operator. It might be said that thrice daily irrigation with hot normal saline solution is sufficient, and the sac should be washed out once daily with a minimum of trauma. This means that introduction of a long canula, or of a canula with sharp ends from which the plating has been worn, should be avoided. Most clinicians seem opposed to the continuance of attempts at probing when dacryocystorhinostomy has been decided upon. For control of pus during the days of preparation, mercurochrome, 2 per cent, or 1/4,000 bichlorid of mercury have been used. Argpyrol and other silver albuminates are avoided in most clinics, due to the fact that in certain cases the fragility of the sac permits easy escape of the silver beneath the skin with disastrous staining. A weak bichlorid ointment is helpful in cases which are prone to blepharitis, and may well be used in all cases for two or three nights prior to operation.

In the nose, especially when dealing with recently healed suppurative conditions, gentle washing with bloodwarm plasma or saline solution may be practiced twice a day. If much force be used, traumatism may result; a swelling of the nasal mucosa which will defeat the whole purpose of the washing. A mild chloretone oil, perhaps 1 per cent, in heavy liquid petrolatum, dropped into the nostrils, will allay a great deal of minor irritation. Chronic infective processes in the nasal

passages may be held somewhat in abeyance by dropping in 2 per cent mercurochrome or 25 per cent argyrol solution.

For immediate preparation the same mercurochrome or 3 per cent tincture of iodine may be painted over the nasal aspect of the region of the sac prior to the opening of the mucosa. However, in view of the careful washing out of the sac itself at the time of operation, and the fact that these antiseptics are often in use during the progress of the operation preliminary intranasal swabbing is not strictly required.

For skin preparation most clinicians seem to prefer gentle soap and water washing, drying and very careful employment of benzine and weak iodine solution. Care must be exercised to avoid conjunctival irritation.

In all these details it may be said that the question of individual choice and the question of the experience of the clinician should have the first influence.

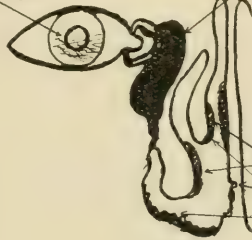
Relative to indications or contraindications, it may well be stated that this is not a procedure to be lightly undertaken; it is not a procedure for a case that may be attacked by easier means. Dacryocystorhinostomy should not be considered except where other means have been carefully tried and found wanting.

Fig. 1.

INDICATIONS FOR DACRYOCYSTORHINOSTOMY.

Recurrent ulcer
and keratitis

General septic absorption
(kidney, heart, bowel)



Chronic nasal suppurations
septal ulcers
ozoena



Pharyngitis lateralis

Recurrent tonsillitis

a. The need for speedy radical cure.

Fig. 2.

Chronic conjunctivitis

Blepharitis marginalis

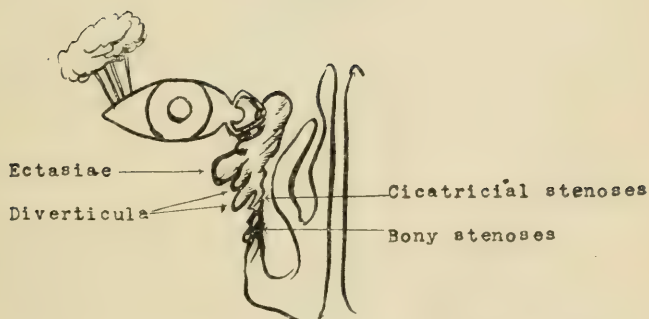
Ulcer serpense

Recurrent phlegmon

Danger of erysipelas

b. The need for a clean eye. (Impossibility of intraocular operations.)

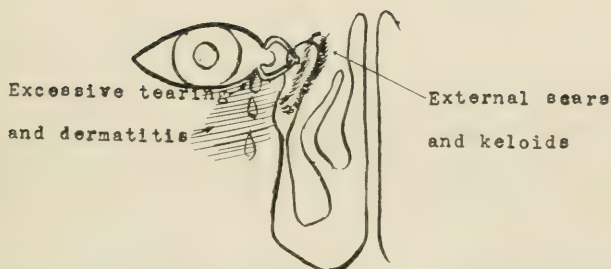
Fig. 3.



c. The need for restoration of physiologic function.

Avoidance of excision of the lacrimal gland.

Fig. 4.

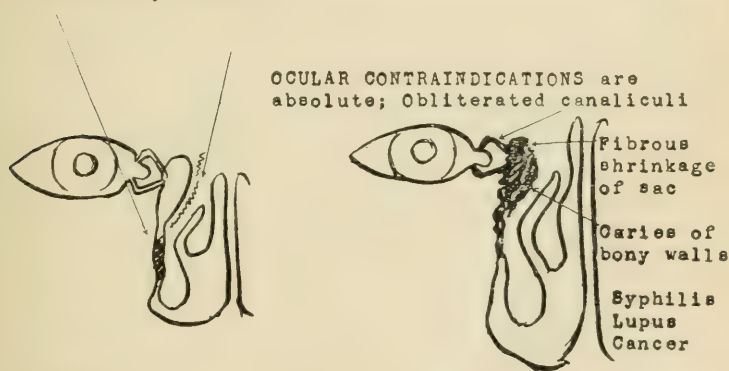


d. The need for good cosmetic results. Avoidance of dacryocystorhinostomy in all its types requires:

1. Nose must be roomy or be made so.
2. There must be one functioning canaliculus.
3. There must be enough of the sac to act as a drainage channel or syphon.

Fig. 5.

GENERAL CONTRAINDICATIONS.—In babyhood, time will take out the occluding débris. In age, palliation is preferable; beware of brittle bony structures.

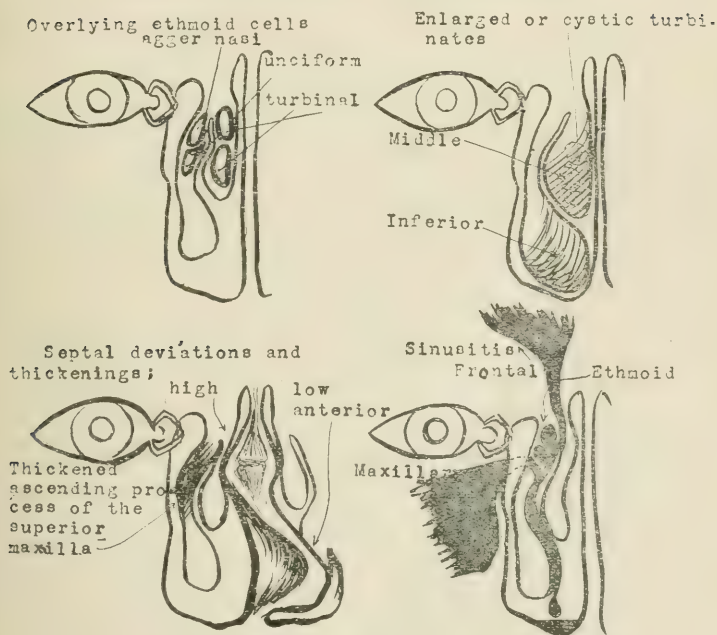


Disease contraindicating general anesthesia; cardiovascular, renal; diabetes; tuberculosis and syphilis.

Keloidal tendencies in negroes; pyodermic or erysipelatous tendencies in others.

Fig. 6.

Nasal contraindications, usually remediable:



VI.

USE OF TUNING FORK STEM FOR BOTH AIR AND BONE CONDUCTION IN THE RINNE TEST.*

BY ROBERT SONNENSCHN, M. D., AND

JOHN P. MINTON, PH. D.,†

CHICAGO.

Since the method of testing the relation of bone to air conduction as first described by Rinne in 1855 is familiar to all, we will not dwell upon the details thereof. When no longer heard via bone the small a^1 fork (435 d. v.) is, according to Bezold, heard via air an average of 30 seconds longer. He found that when the fork is held only at the meatus without having been previously placed on the mastoid, the duration of air conduction is longer than when the usual Rinne test is performed, i. e., the small a^1 fork (435 d. v.) was heard 70 to 80 seconds.

In another paper Bezold calls attention to the fact that when the Rinne test is carried out in the usual manner and the fork is no longer heard with the prongs held near the meatus, if the stem be then inserted into the auditory canal, the fork is again heard via air (?) and usually for a period of about 12 seconds. Since pressure of the stem against the meatal walls tends to diminish the duration of the fork's vibration, even this test does not show the full extent to which a fork may sound. Bezold claims that "we could really determine the length of hearing by air conduction only if we were able to approach the drum membrane as closely with the prongs as we do with the stem of the fork."

*Read at May 8, 1922, meeting of the Chicago Laryngological and Otological Society.

†The work of Dr. Minton has been done under the auspices of the National Research Council as a National Research Fellow in Physics at the Ryerson Physical Laboratory of the University of Chicago.

In a short paper entitled "A Suggestion Regarding the Rinne Test" read before this society February 16, 1915, one of us (Sonnenschein) analyzed the results found in one hundred ears in which the stem of the fork was placed in the meatus auditorius externus after the bone and air conduction had been determined in the usual manner.

It was found that the fork was then heard via meatus an average of 13 seconds longer than with the prongs held near the ear. In the pathologic cases (fifty ears) the average was 10.7 seconds; in the normal cases (fifty ears) the average was 15.2 seconds. Our concluding query was that, while the fact of this prolonged hearing of the fork may prove to be more academic than practical in value, should we not apply a routine which requires but a trifling amount of extra time and may in the end bring forth enlightening data?

We have always felt, however, that placing the fork stem in the meatus did not give hearing only via air, since the contact with the tissues permitted some transmission via cartilage and bone.

In a paper entitled "A Study of the Rinne Test in One Hundred Cases" (accepted as thesis by the American Rhinological, Laryngological and Otological Society, 1921) one of us (Sonnenschein) calls attention to the fact that Boenninghaus quotes Zimmermann and Quix, who deny all value to the Rinne test because it measures the difference between two factors, namely, the vibration of the fork stem and its prongs, which are physically so different that they are not comparable. Others have also raised the same objections to this test. Boenninghaus admits this, but says that since we make the same error in each test, the results can be compared with one another, and that suffices for the practitioner, even if it does not for the physicist.

Conceding the viewpoint of Boenninghaus, which is supported by the practical experience of innumerable otologists, that the Rinne test as usually performed gives us a ratio between bone and air conduction which furnishes us with most valuable information, it seemed to us desirable to demonstrate, if possible, that air conduction was feasible by means of the fork stem. In that case the stem would be employed for both

bone and air conduction factors of the Rinne and the theoretical objections of Zimmermann and others overcome.

While the prongs move in transverse vibrations of large amplitude, the stem at the same time and for just as long a period shows longitudinal vibrations of smaller amplitude. It is desirable from the acoustic viewpoint to test both air and bone conduction by the same type of vibrations such as those given off by the stem of the fork. The present method of testing air conduction by forks is subject to a serious objection because of the disturbance of the intensity of the sound surrounding the prongs of the fork. The prongs, as is well known, move in opposite directions, so that when a condensation and a rarefaction are propagated outward these very nearly but not exactly overlap, with a resulting interference with the sound. There are distinct surfaces of maxima and minima of sound, as illustrated in the accompanying sketch. The plane surfaces (1 and 2) represent the maxima, while the curved surfaces (3 and 4) represent the minima of sound intensity. Thus the intensity of the sound changes greatly with slight rotation of the fork about the vertical axis in the figure. Not only does the intensity change about the vertical axis, but it also changes in a modified manner at various distances from the stem. In addition to these factors, there is another one of great importance, namely, the slippage of air molecules around the prongs of the forks, which varies with the amplitude and velocity of motion of the prongs and causes a further modification of the sound intensity about the fork. Moreover, all of these factors vary with the pitch, so that it is evident that air tests made in the usual manner are subject to much variation in precision. There can be no such variation as this of the sound intensity in the air surrounding the stem of the fork, and for this reason tests of air conduction made with the stem in the manner reported herein are not subject to these objections.

Anticipating what is to follow, it may be noted that there is little loss of sound energy through the walls of the rubber tubing into which the stem of the fork is inserted. Nearly all of the sound is transmitted along the air column, and little if any at all is propagated by molecular conduction along the wall of the tube.

The question of the length of the tubing is therefore not of great physical importance, and its decision may be left to each individual otologist, with the proviso that not too wide a tube be used, lest it act as a resonator (a diameter of from 0.5 to 0.8 cm. is recommended).

Although a search of the literature was made, no reference to the modification of the test as done in the manner to be described was found.

(Note.—Since the reading of this paper an abstract of an article by Stefanini of Turin was found, in which he calls attention to another method of testing both bone and air conduction by means of the fork stem.)

"The handle of the fork is attached to a disc of wood, to the bottom of which are affixed two pieces of metal of equal length and 4 mm. in diameter. To one of these is attached a rubber tube 1.4 meters long. The other metal strip is placed on the skull, and its vibrations are transmitted through bone, while the rubber tube is introduced into the ear, and the vibrations from that strip of metal are transmitted through the air, the vibrations in both cases having the same source." Stefanini also believes if the tragus is pushed into or across the meatus and the fork stem placed thereon that the sound is conducted by air. Gradenigo thinks this holds true only in normal, not in pathologic ears.

The method employed is to excite the fork in the manner chosen by each individual otologist endeavoring to do this in a definite way in order to get uniform results. We used the small a^1 fork (435 d. v.) of Bezold. Holding this at right angles to the body, a small pleximeter (such as employed by neurologists for testing reflexes) is allowed to drop of its own weight from a perpendicular to a horizontal position, striking the flat surface of the prong. The stem is then held against the mastoid process in the usual manner and the duration of hearing by bone conduction noted. The fork is then inserted into one end of a rubber tube, the other end of which is attached to a vulcanized rubber olive shaped tip (such as often is used with a Politzer bag) and the latter inserted into the meatus auditorius externus, care being taken that the lumen of the tip is not occluded by contact with a part of the

concha or wall of the meatus. The duration of hearing via air conduction is then noted.

The dimensions of the tubing used were: Length, 51 cm.; width of lumen, 0.7 cm., and thickness of wall, 1.5 mm. Clinically this method of doing the Rinne test was carried out on 35 cases. First the test was performed in the usual manner and then repeated by means of the rubber tubing as described.

N. B.—The sign + before the Rinne result means that it is positive—that is, air conduction longer than bone conduction. The sign — means that the Rinne is negative, namely, bone conduction is longer than air conduction.

Age, History, Diagnosis, etc.	Membrana tympani	Whisper	Weber	Schwabach	Rinne	Modified Rinne
Case 1—Age 6, F. Otitis Media. Sinistra acuta.	Right normal. Left infected and desquamating.	Rt. 5 m. + Lt. 0.5 m.	Left	Length	Rt. bc 32 ac 30 Lt. bc 44 ac 30	ac 25 ac 20
Case 2—Age 48, F. Chronic tubal catarrh with some nerve degeneration, dextra.	Dullness and retraction, especially right.	Rt. 3 m. Lt. 5 m. +	Right	Length	Rt. bc 25 ac 30 Lt. bc 25 ac 38	ac 28 ac 38
Case 3—Age 27, M. Auditory nerve degeneration, sinistra.	Slight dullness; otherwise negative.	Rt. 4 m. + Lt. 0.6 m.	Right	Much shortened	Rt. bc 18 ac 30 Lt. bc 10 ac 20	ac 40 ac 40
Case 4—Age 27, M. Germen and very early auditory nerve degeneration.	Dullness.	Rt. 5 m. + Lt. 5 m. +	Left	Shortened	Rt. bc 18 ac 30 Lt. bc 30 ac 30	ac 40 ac 40
Case 5—Age 25, M. Otitis media suppurativa dextra chronica.	Left drum dull. Right membrane almost gone. Pus.	Rt. 20 cm. 4 m. Lt. 7 m. 2 m.	Left	Increased	Rt. bc 22 ac 15 Lt. bc 15 ac 15	ac 20 ac 18
Case 6—Age 69, M. Some nerve degeneration with middle ear.	Much retraction both sides with dullness of right.	Rt. 0.3 m. Lt. 3 m.	Right	Shortened	Rt. bc 25 ac 18 Lt. bc 20 ac 15	ac 15 ac 20
Case 7—Age 52, M. Tubal catarrh dextra with slight nerve change.	Slight dullness both drums.	Rt. 5 m. + Lt. 5 m. +	0	Shortened	Rt. bc 20 ac 25 Lt. bc 15 ac 35	ac 35 ac 40
Case 8—Age 22, F. Chronic tubal catarrh.	Slight brown discoloration.	Rt. 4.5 m. Lt. 3 m.	0	Lengthened	Rt. bc 28 ac 25 Lt. bc 25 ac 30	ac 24 ac 25
Case 9—Age 27, F. Tubal catarrh.	Some dullness.	Rt. 5 m. + Lt. 5 m. +	Left	Normal	Rt. bc 18 ac 28 Lt. bc 15 ac 20	ac 32 ac 30

Age, History, Diagnosis, etc.	Membrana tympani	Whisper	Weber	Schwabach	Rinne	Modified Rinne
Case 10—Age 55, F. Tubal catarrh with some nerve degeneration.	Slight refraction left.	Rt. 1.5 m. Lt. 3 m. +	Left	Shortened	Rt. bc 20 ac 25 Lt. bc 17 ac 32	ac 32 ac 30
Case 11—Age 16, M. Beginning otosclerosis (?).	Slight brownish discoloration.	Rt. 4.8-4 m. + Lt. 4.8-2.5	Right	Lengthened	Rt. bc 30 ac 30 Lt. bc 25 ac 30	ac 25 ac 25
Case 12—Age 38, F. Slight tubal catarrh.	Drums dull.	Rt. 5 m. Lt. 5 m.	0	Normal	Rt. bc 20 ac 35 Lt. bc 20 ac 35	ac 50 ac 50
Case 13—Age 46, F. Auditory nerve degeneration right.	Fairly negative.	Rt. 0.3 m. Lt. 4 m.	0	Increased	Rt. bc 20 ac 10 Lt. bc 15 ac 20	ac 10 ac 28
Case 14—Age 48, M. Nerve degeneration with tubal catarrh right.	Some refraction and dullness, especially right.	Rt. 20 cm. Lt. 5 m.	Right	Increased	Rt. bc 35 ac 20 Lt. bc 35 ac 35	ac 15 ac 32
Case 15—Age 64, F. Beginning nerve degeneration with some tubal catarrh.	Dullness and some cloudiness.	Rt. 2 m. + Lt. 3 m. +	0	Diminished	Rt. bc 16 ac 20 Lt. bc 12 ac 25	ac 25 ac 35
Case 16—Age 46, M. Beginning nerve degeneration.	Right drum dull.	Rt. 6.7-2.0 m. Lt. 5 m.	0	Diminished	Rt. bc 18 ac 20 Lt. bc 16 ac 20	ac 25 ac 18
Case 17—Age 22, F. Otosclerosis (?).	Drums dull.	Rt. 1.3 m. Lt. 0.3 m.	Right	Lengthened	Rt. bc 40 ac 30 Lt. bc 40 ac 20	ac 25 ac 18
Case 18—Age 61, F. Otitis media chronica (sin.). Nerve degeneration and chronic tubal catarrh.	Large dry central perforation, right drum retracted. Left somewhat dull and atrophied.	Rt. 0.3 m. Lt. 20 cm.	0	Shortened	Rt. bc 25 ac 22 Lt. bc 25 ac 25	ac 35 ac 25

Age, History, Diagnosis, etc.	Membrana tympani	Whisper	Weber	Schwabach	Rinne	Modified Rinne
Case 19—Age 49, F. Auditory nerve degeneration right.	Some dullness.	Rt. 10 cm. Lt. 5 m. +	Left	Shortened	Rt. bc 8 ac 10 Lt. bc 15 ac 35	ac 10 ac 35
Case 20—Age 22, F. Otitis media chronica (dextra). Otitis externa (dextra).	Right drum infected. Small perforation antero-inferior quadrant. Left drum negative.	Rt. 5 m. + Lt. 5 m. +	Right	Normal	Rt. bc 18 ac 25 Lt. bc 17 ac 35	ac 28 ac 35
Case 21—Age 26, F. Otitis media chronica.	Left drum retracted. Perforation antero-inf. quadrant right drum.	Rt. 3.5 m. Lt. 5 m. +	0	Shortened (?)	Rt. bc 20(?) ac 30 Lt. bc 25 ac 24	ac 30 ac 24
Case 22—Age 53, M. Tubal catarrh chronic.	Retracted and dull drum membranes.	Rt. 1.5 m. Lt. 5 m. +	Right	Shortened (?)	Rt. bc 27 ac 25 Lt. bc 18 ac 35	ac 31 ac 38
Case 23—Age 40, M. Tubal catarrh and some nerve degeneration.	Drum membranes fairly negative.	Rt. 1.5 m. Lt. 2 m.	0	Shortened	Rt. bc 15 ac 20 Lt. bc 15 ac 15	ac 20 ac 20
Case 24—Age 48, M. Chronic otitis media with nerve degeneration.	Right scar near center. Left dull.	Rt. 20 cm. Lt. 1 m.	Right	Shortened	Rt. bc 12 ac 0 Lt. bc 15 ac 30	ac 0 ac 0
Case 25—Age 61, F. Otitis media chronica (sin.). Nerve degeneration with chronic tubal catarrh, right.	Large dry central perforation left, with retraction right drum and atrophic.	Rt. 0.3 m. Lt. 20 cm.	0	Shortened	Rt. bc 25 ac 22 Lt. bc 25 ac 25	ac 35 ac 25
Case 26—Age 52, F. Tubal catarrh.	Dullness both membranes.	Rt. 4 m. + Lt. 4 m. +	Right	Increased	Rt. bc 24 ac 28 Lt. bc 20 ac 38	ac 33 ac 42
Case 27—Age 41, F. Normal ears.	Negative.	Rt. 5 m. + Lt. 5 m. +	0	Normal	Rt. bc 24 ac 35 Lt. bc 22 ac 35	ac 40 ac 40

Age, History, Diagnosis, etc.	Membrana tympani	Whisper	Weber	Schwabach	Rinne	Modified Rinne
Case 28—Age 48, F. Normal.	Negative.	Rt. 5 m. + Lt. 5 m. +	0	Normal	Rt. bc 20 ac 40 Lt. bc 22 ac 43	ac 40 ac 43
Case 29—Age 23, F. Normal.	Negative.	Rt. 5 m. + Lt. 5 m. +	0	Normal	Rt. bc 25 ac 45 Lt. bc 25 ac 45	ac 45 ac 45
Case 30—Age 29, M. Normal.	Negative.	Rt. 5 m. + Lt. 5 m. +	0	Normal	Rt. bc 25 ac 50 Lt. bc 25 ac 50	ac 60 ac 60
Case 31—Age 30, M. Normal.	Negative.	Rt. 5 m. + Lt. 5 m. +	0	Normal	Rt. bc 18 ac 30 Lt. bc 20 ac 40	ac 35 ac 50
Case 32—Age 60, F. Otitis media chronica sinistra.	Refraction right membrane. Much destruction left.	Rt. 3 m. + Lt. 1.5 m.	0	Increased	Rt. bc 25 ac 28 Lt. bc 24 ac 22	ac 36 ac 26
Case 33—Age 54, F. Otitis media acuta (dextra), healed.	Drum membrane negative except slight injection of right.	Rt. 2.5 m. Lt. 5 m. +	Right	Normal	Rt. bc 20 ac 18 Lt. bc 15 ac 20	ac 20 ac 35
Case 34—Age 25, F. Otitis media acuta (sinistra).	Left drum rod. Small perforation.	Rt. 5 m. + Lt. 0.6 m.	Left	Normal	Rt. bc 35 ac 40 Lt. bc 30 ac 25	ac 45 ac 35
Case 35—Age 18, F. Normal ears.	Negative.	Rt. 5 m. + Lt. 5 m. +	0	Normal	Rt. bc 22 ac 30 Lt. bc 22 ac 35	ac 40 ac 40

N. B. In the above table Weber designated as zero means no lateralization. Right or left refers to the side on which fork is best heard. Since examining room was only about 5 m. long, the hearing is usually designated as plus where 5 m. is noted, since the hearing was greater than the distance at which tests could be made.

Of these cases, 6 had normal ears, 12 showed either tubal catarrh or acute or chronic otitis media, one was apparently a typical otosclerosis, one was suspicious of a beginning otosclerosis, 5 showed auditory nerve degeneration and 10 apparently had both middle and internal ear changes.

ANALYSIS OF CASES.

We see that in 23 cases the air conduction in both ears was from 3 to 15 seconds longer when the fork was connected with the tube. This group included 4 normal ears, 5 cases of nerve degeneration, 8 combined middle and internal ear cases, and 6 cases of pure middle ear affection. In 2 cases (one normal, the other a chronic otitis media with some nerve change), the hearing in both ears via tube was the same as when the prongs were held near the ear in the usual manner. In 5 cases, only one ear showed the same length of hearing by both methods. The fork was heard a shorter time with tubing than with the prongs of the fork by from 2 to 10 seconds in 9 cases, of which 2 were moderate nerve degenerations, one a combined middle and internal ear involvement, 4 showed either tubal catarrh, acute or chronic otitis media, and 2 were the otosclerosis cases before mentioned.

The fact that some cases with very distinct middle ear affection and a negative Rinne showed less hearing of the fork via tubing and tip in ear than when the prongs were held near ears seems to show that in the method we have used the hearing of the stem is by air and not by bone conduction; otherwise in these very cases it ought to be increased just as it is when the stem is held against the mastoid process. Objections might be raised to this method of testing air conduction, such as the fact that it is the wall of the tube or hard rubber tip which transmits the sound waves to the cartilage or bone of the meatus and surrounding parts just as when the rounded end of the fork stem is inserted directly into the meatus.

It can, however, be demonstrated that sound waves are transmitted almost entirely through the confined column of air within the tube and not by the walls thereof. Molecular vibrations, however, may be conducted by the tube wall, but the following experiments will show that no such vibrations are present in these tests and that what is heard is entirely by

air conduction. When tubing is clamped in a vise (a length of about 10 cm. being compressed) the sound of the fork is not heard via tube lumen. If the fork is placed on the outside of the tube the sound is heard if the fork rests between the vise and the ear, but if it is put on the farther side of the tubing—i. e., beyond the vise—nothing is heard via tubing. If the rubber tube is cut and the sections held in proximity but not in contact with one another the sound of the fork is heard via tube lumen; but when section is clamped in the vise there is no transmission of sound. When a steel tube is placed in the vise and the fork rests on it, the sound is transmitted, since the lumen, of course, is not compressed. If a steel rod 8 or 10 cm. long is inserted into the ends of the tube sections the sound of the fork is not heard via lumen of the tube. Plugging the opening in the olive hard rubber ear tip with wax, wood, etc., merely reduces the intensity of the sound heard. That a little of the sound is transmitted through but not along the tube wall to the outside is shown by the fact that when the tubing or olive tip are held tightly against the auricle a faint sound only is heard. When thus held, however, and with the olive tip open, the sound is heard, thus showing that the sound is transmitted to the ear through the open end and only slightly through the wall of the hard rubber olive tip.

From the clinical standpoint we believe that the results obtained in a number of cases show definitely that the fork when used with tubing and tip in the ear is not heard via bone but only via air.

In several cases, presumably with otosclerosis or some other middle ear involvement, where a prolongation of bone conduction, either relative or absolute, would be expected, we find, as previously noted, that with the tip in the ear the fork is heard a shorter time (i. e., fork on mastoid) than were the prongs heard via air.

Permit it to be said that in presenting the results of this little investigation there has been no intention of discrediting in any degree whatever the Rinne test, which gives the otologist such valuable information, but merely to show that the stem of the fork may be used for determining the duration of both the bone and air conduction.

CONCLUSIONS.

1. Physical experiments show that with the fork stem inserted in the end of a rubber tube the sound waves are transmitted to ear by air column and not by way of the tubing wall.

2. Clinical findings show that with fork stem in tubing connected with the ear via hard rubber olive tip, the sound is in many cases heard somewhat longer than with prongs held near the ear. In most cases, however, of middle ear disease, especially those with negative Rinne, the hearing via tube was somewhat less than that with the prongs, whereas if the hearing had been via bone conduction it should have been increased with the tip in the ear.

3. It therefore seems feasible to determine not only bone conduction as is usually done, but also the air conduction in the Rinne test by means of the stem of the fork.

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29 E. MADISON ST.

VII.

AURAL EXOSTOSIS.*

BY FRANK A. BURTON, M. D.,

DENVER, COLO.

Although aural exostoses are known to have been fairly common centuries ago, history fails to record any effort to obtain comprehensive knowledge of the affection until as recently as 1877, when J. P. Cassells in the *British Medical Journal* claimed "to be the first observer who has given a rational explanation of aural exostosis, supported by clinical and pathologic facts." While in recent years some advancement in scientific knowledge of this disease has been made, and while many views have been given as to the cause of the condition, its etiology is still largely speculative. On review of the literature one finds that the only etiologic factors upon which the majority seem to agree are that of the influence of heredity and that of some forms of local chronic irritation, noninflammatory or inflammatory. As in otosclerosis, while the anatomic changes of the disease are quite well known, the pathogenesis is still in the realm of uncertainty. Therefore one ventures to think that the imagination may be given full play. It is interesting to note that the hypotheses advanced as to the etiology of otosclerosis are practically identical with those suggested in exostosis of the aural canal. According to Gray, exostoses are not infrequently found in the bony labyrinth. He has reported cases of otosclerosis which at autopsy showed exostoses in the region of the promontory, oval and round windows. He quotes Katz as having recorded a case in which the hammer and the anvil suffered. Fischer found exostoses on the stapes in this connection also. The writer ventures to think that aurists making a comparative study of the two conditions will be, as he is, impressed with the belief that the two processes differ fundamentally in the point of

*Candidate's thesis. American Laryngological, Rhinological and Otological Society.

location only. Later on in the paper this statement will seem less radical. There is abundant evidence that this disease was once very frequent among primitive races. In reviewing the literature and contrasting reports of prehistoric specimens with those of modern times, one can but be convinced that in the dim and misty past there were etiologic factors which exist now in part only. To review the literature, present a case, report a comparatively large number of unreported exostoses in prehistoric specimens and to postulate what seems to the writer a logical theory as to the fundamental cause of the condition, is the purpose of this paper.

In going through the literature of this subject, one is greatly confused by the lack of uniformity or system in the terminology. Each author has a different nomenclature or none at all. Should not "canal" be used instead of "meatus"? Would it not be better for "exostosis" to take the place of "hyperostosis" and "osteoma" until a definite pathologic basis for a classification is agreed upon by all? By Kerrison, Blake and Field, all bony growths of the canal are considered "exostoses," while Politzer, Peters, Ellis, Moss, Connal, Davis, Lake, Green and Hartman use the more complicated terminology. Ballenger says that hyperostoses and exostoses are often spoken of synonymously, although according to strict pathologic interpretation they should be used to describe different lesions of bony tissue, the former being the diffuse thickening of the wall. Pritchard and Blake regard the hyperostoses as resulting more particularly from chronic inflammation, while Field speaks of the general thickening of the wall as the rarest form of exostosis. Sheppard reports two cases of "osteoma" and Ballance one "of the temporal bone blocking the outer part of the external meatus."

As in terminology, so in the matter of etiology there is great diversity of opinion, in some instances views of equally qualified otologists being diametrically opposed. All references credit these growths with being single, multiple, unilateral or bilateral, and in form broad based, flat or pedunculated, some even without a bony attachment (Schumacher, Jacquemant and Delstanche). In frequency, Blake places them at five per thousand, Sheppard at four to five, and Jackson at eleven per three thousand cases of aural disease, showing a compar-

ative rarity today. Green believes they occur in fetal development, youth or early adult life. Dalby said they were congenital. Kerrison quotes Bezold, believing that the condition is rarely or never developed before puberty, but in contrast Hartman states that their growth terminates with the growth of the individual. As to the point of origin and location, Cassells believed that they always arise from the mastoid periosteum of the outer half of the canal and always from the posterior wall; Pritchard and Blake, on the bony walls or the outer edge of the osseous meatus; Cassells and Pritchard again, and Ballenger, at the junction of the osseous and the cartilaginous canals. Kerrison, Blake, Jackson and Green have found them most often on the anterior or posterior walls—if single, on the anterior near the drum. Politzer alone mentions their existence on the floor or inferior meatal wall, and Ballance has published the only reference to one on the roof or superior wall—i. e., Toynbee's case, and yet Cheattle limits them to the tympanic portion of the temporal bone. In point of location in the canal, Green places hyperostoses farther out than exostoses, and reports a case of exostosis in which the osseous canal was missing, except the upper and lower posterior portions, the growth being attached to the tympanum or attic. He also reported two other cases of bony growth located just within the meatus originating from the cartilaginous portion of the canal, and Lichtenberg one also.

Cassells believed that these growths are due to a rupture of a subperiosteal abscess into the canal at the junction of the cartilaginous and bony portions, or through the cartilage, discharging pus, and that the exostosis resulted from ossification of the granular growths that spring up from the abscess opening. St. John Rossa in 1866 published four cases of "true exostosis," "morbid growths consequent on local irritation," in three being pus from the middle ear. Cocks reported a case showing bony metamorphosis in two polypi, in the course of an O. M. P. C. Pritchard held that one type of multiple exostosis originates in this manner, but Weydner reported thirteen aural polypi examined microscopically and says that he has never been able to discover new bone formation in any that he has examined. Knapp definitely states that the pathogenesis is this—O. M. P. C., periostitis, polypi, and finally

exostosis, using a case of 34 years' otorrhea as an illustration. Ballenger, Politzer and Jackson believe the condition may be due to a fracture of the bony canal and the resulting periostitis or a chronic middle ear suppuration might excite a secondary inflammation of the membranous canal, causing a fibrous or connective tissue thickening which after a long period of time might be metamorphosed into osseous tissue. Green believed them to be the result of a circumscribed periostitis and found only in adults rather than youths. Carmalt regarded only the fibrous covered exostoses as inflammatory in origin, the cartilaginous being otherwise. McBride has reported a case of exostosis filling the canal, with a history of repeated attacks of inflammation, and Dalby two associated with O. M. P. C. Lucae's patient had otorrhea for 23 years and the canal was practically occluded by two bony growths. One patient of Toynbee's suffered from a foul discharge for 20 years, and he stated that in 1013 diseased ears examined, 14 showed narrowing of the external meatus as a result of bony tumors from the osseous walls.

In the opinion of Field, aural exostoses are caused by some local source of irritation, in many cases this being irritation by salt water repeatedly introduced through protracted bathing. Two of his 27 cases gave sea bathing as a cause. Dalby, Pritchard and Jackson concur in this opinion, and the latter quotes Koerner, 147 cases, with those living by the sea most affected. Connal states that this condition is more prevalent in England than on the continent and extremely frequent among the South Sea Islanders. Ballance has reproduced two cases, one of a Chatham Islander, and one of a Melanesian Islander (Welcher), but states that "constant bathing in the sea is not the sole cause of these growths." Exostoses of the external meatus often occur in those born in former German Africa, as well as in the German troops stationed there—a residue of a chronic ossifying periostitis of the meatus endemic to that country (Müller). These possessions are on salt water. Blake is opposed to the aquatic theory, mentioning the fact that the mound builders, a large percentage of whom are known to have had exostoses, lived inland.

Ballenger and Pritchard believe that in some cases syphilis may be a factor, although not to a great extent. By Blake,

too, quoting Toynbee and Gruber, syphilis is given a very meager role. Jackson regarded this disease as seldom if ever the cause, and Field denies any conclusive evidence at all as to the luetic origin. Ballenger and Field state that gout is thought to be another cause, but say it is doubtful if this leads directly to their formation. Pritchard and Connal believe that rheumatism may be added to the list. In Blake's list, again referring to Toynbee and Gruber, both gout and rheumatism are found. Their syphilitic, gouty or rheumatic relation can only be held valid when other osseous tumors occur simultaneously in other parts of the body, the origin of which can be traced to the original disease (Politzer). By Kreible and Bergeim, a metabolic study of a case of multiple exostosis was made with the result that the findings did not support the view of a relationship of this disorder to rickets or "to any primary disturbance of the general metabolism."

Heredity is listed by Ballenger as an etiologic factor, saying that this condition may be traced through a few generations. Dalby saw exostoses in the aural canals of a father and two sons and regarded them as congenital in some cases. Politzer quoted Schwartze regarding a hereditary predisposition as a factor, having seen such cases several times. Hartman reports a mother and sister having the same condition, and quotes Boyer, who reported a female whose father, brother, three sisters, two nephews and all her own children also were affected. He also exhibited two specimens of hyperostosis and held them to be anomalies of development for the reason that (a) they appeared simultaneously in both ears, (b) that they were limited to the pars tympanica, (c) that there was a uniform consistency of the rest of the bone. Lake denies Hartman's theory, but Politzer regards some cases as arising during the stage of development and ossification. Green, while mentioning a case of Jacks, reports three "true osteomata" of the mastoid, which he believes were probably of slow noninflammatory growth, probably fetal in origin, originating in the tympanum or close to it. All were covered with normal skin. In two the osseous portion was overlaid with cartilage, and in one with fibrous tissue. In the discussion, Carmalt expressed his belief that the cartilage covered ones were fetal in origin and the other inflammatory. Whitney ascribed them

to fetal cartilage rests. Peters had a case in which there was prominence of the supraorbital and temporal ridges, suboccipital ridge marked, and hyperostoses of the nasal bones as well as of the lower jaw anterior to the masseters. In the discussion of this case, Jones suggested that general hyperostosis of the skull might be a stigma of degeneration. Politzer states that in many instances the cause is not ascertainable at all, because the development is not accompanied by inflammatory phenomena, and we are seldom in a position to observe their growth clinically. In view of all this confusion as to the causation we may hide behind Virchow's statement that the etiology of these growths is an irritative disturbance of normal bone development. This "irritative disturbance" may be of any kind or variety, but the underlying question of why the condition arises in some and not in others with a similar aural disturbance cannot be answered with certainty until the broad question as to the etiology of all new growths, malignant or benign, has been answered. The balance in cell growth is in some way upset by a vasotrophic element or an irritative one or by some factor which in many cases may be of such a nature that no history can be elicited. Naturally in a case that we may say is idiopathic or spontaneous in origin, the patient can give no history except perhaps one pointing to a general bodily condition which may be present.

Of historic interest in treatment are two cases refusing operation that Barr improved in respect to hearing, by providing a narrow chink through the canal by counter-irritation behind the ear, leeches on the tragus, and iodine liniment. Dental burrs, snares, metal rods and laminaria tents have been advised, the dilatation method being that of the early aural surgeons, von Trölsch and Bonnafont. Politzer recommends operation for removal of (a) marked deafness due to complete closure exits, (b) marked deafness with disturbance of hearing in the other ear, (c) inflammation in the external ear with interference with drainage, doing a radical mastoidectomy if the condition is complicated with an O. M. P. C., otherwise the gauge and mallet through the external canal. Goldstein interferes surgically for (a) pain from mechanical pressure, (b) obstruction to free drainage, (c) serious impairment of hearing. Field believes they should be removed

only if they interfere with free drainage; Connal also. Powell advises a mastoidectomy in case of middle ear suppuration, but suggests that one should not operate in cases of exostosis with a broad base because of the fear that growth might extend into the tympanum. The chance of benefiting the patient should not be rejected because of this possibility, according to West. Selfridge in a personal communication referred to a case of his own in which it was necessary to do a bilateral modified radical operation after many minor operations had been tried. Finally one ear was converted into a radical mastoid on the advice of Duel and of Hammond, owing to the formation of an exostosis in contact with the annulus.

With the view of laying a further foundation for part of my conclusions, the development of the osseous canal has been studied. According to Kerrison, firm osseous union of the annulus with the rest of the temporal bone occurs during the first year of life, the free ends of the ring having united with the squama about the time of birth (Spalteholz). By deposition of new bone upon the outer surface of the tympanic ring, the annulus is converted into a bony trough, citing the fact that the sulcus tympanicus remains unchanged at the inner margin of the canal as proof that the conversion of the tympanic ring into a partial canal is effected solely in this manner. In Quain's Anatomy one reads that the tympanic element begins in the form of an incomplete ring to which the membrana tympanica is attached. On the outer side of the ring is a fibrous plate occupying the same position in the wall of the meatus as the future bony plate. Ossification extends from the annulus into this fibrous tissue. Hertwig-Mark, in their textbook, call attention to the fact that the annulus gradually becomes broadened into a bony plate which fuses with the petrosal bone. This growth does not occur uniformly from the ring, but mainly from two tubercles situated one near the upper end of its anterior limb, and the other at the lower posterior, so that the outer edge of the tympanic bone at this stage exhibits a notch which by the subsequent union of the two tubercles is converted into a foramin. This aperture occasionally persists until adult life, and in cases where it is closed, the osseous wall remains thin. While these changes are forming an osseous wall in front, below and be-

hind, the squamous part of the temporal bone situated above the membrana tympanica grows outward and comes to be nearly at a right angle with the vertical plate of the squamosa so as to form a roof. This process of formation is usually completed during the second year. Minot simply states that "the tympanic ring is incomplete on the lower anterior side and remains so for several years after birth." To illustrate the process of fetal development, the specimens in the San Diego Museum, Anthropological Department, have been photographed and the pictures with some descriptive matter are here presented.

All reports point to the origin of these growths from the anterior and posterior walls—practically never from the floor or roof (only one or two exceptions). Disregarding the many theories as to the direct cause, may not one assume that if the exciting agent be some form of chronic irritation and the potentialities of all four walls be equal, the most dependent part, the floor, would be the wall receiving the maximum stimulation, and hence should be the area most frequently productive of these overgrowths? But since the floor is almost never involved in the process, one seems justified in assuming other etiologic factors than chronic irritation. It seems logical to believe that the exciting agent, whatever its form, acts equally upon the entire canal, but is responded to only by those points especially susceptible to the stimulus. In the vast majority of cases reported only those areas responded to this stimulation, whatever it was, which during the development of the canal grew most rapidly. These places, as has been shown by the embryologic study, are the two ends of the annulus from which the osseous canal proliferated the more rapidly into the fibrous anlage. May not these areas have retained the potentiality of more rapid development and response to any production stimulus? The answer "yes" to this question is the writer's theory as to why the vast majority of aural exostoses are found along the suture lines between the canal and the rest of the temporal bone.

Ever since examining with Dr. Politzer his unusual collection of exostoses of the aural canal, I have been looking for a case. During this period three examples of the process have

come under my personal observation. One has been of special interest to me and is as follows:

Mr. P., age 39, Scotch-Irish, married, five children.

C. C.: Impaired hearing, almost totally deaf; mild tinnitus; sometimes very dizzy; paracusis Willisii.

Family history: Father died of "bleeding piles." At age of forty had a "growth like gristle" removed from the right mastoid. Growth had been growing rapidly enough to cause annoyance. Brother died of "cancer." No history of family deafness could be gotten.

Past history: When the patient was seven months in fetal development his mother was thrown from a horse; hemorrhages of several days' duration followed and emptying of the uterus was advised. Operation refused and pregnancy continued to full term. At birth he "looked like he had no blood at all" and continued weak and anemic until about the age of five. Measles, but no ear involvement.

At the age of two he fell, striking the back of his head, and was unconscious for almost an hour and the same night had a "spasm." These continued every few days until the patient was about five. Since then he has been entirely well.

At the age of eight he began to swim in fresh water. At thirteen he moved to San Diego and since then has swam in salt water, practically "living in the water," as he says. At fourteen he began high diving, from forty to sixty feet, continuing as a professional high diver until 1920.

"Sciatica" often. No positive venereal history.

P. I.: At age of sixteen, noticed that he was having difficulty in clearing his ears of water after diving. This gradually became worse until the sensation of an unremovable obstruction with terrific buzzing in the left early nearly "drove him wild." The buzzing in the right ear began in 1915, just before he came to the San Diego County Ear Clinic. The impairment of hearing in the left ear began about the age of 19. In his attempts to remove the accumulated water from the ears he would often cause bleeding and swelling of the external canals. The canals have always been rather insensitive, with no wax except a few dry light brown scales for the past ten or twelve years. Never any pain or earache. Hears high-

pitched voices better than the low. Canals have been lanced previously, diagnosis of "furunculosis."

Examination: Both canals closed externally by hard, insensitive, immovable growths covered by apparently normal integument. Hears only shout, and the watch on contact, both ears. A. D.: C negative, C² heard faintly, C⁴ heard, Galton O. K. A. S.: C negative, C² negative, C⁴ heard, Galton O. K. Weber to left. Rinne negative, both ears. Schwabach prolonged, both ears. Slight transitory improvement on inflation. Depletion of the canal walls so that a Yankauer eustachian applicator with a bit of cotton on it could be passed caused a marked improvement in the hearing.

At the same time the patient complained of susceptibility to head cold, alternating nasal stenosis with especial disturbance on the right, postnasal dripping, frequent hawking and a dry, irritable throat. Nasal examination showed an edematous mucosa, with a deviated septum in contact with the right middle turbinate body. Slight impairment of translucency of the right antrum.

Operation on the ears was refused, but a submucous resection was agreed upon. Resection done in the clinic in 1915, with relief of the nasal symptoms, except some slight basomotor disturbance at times.

Between this time and 1920 the patient came at intervals of a few months to have his canals treated. Finally, in August, 1920, in the hope for relief from his increasing deafness and the unbearable tinnitus, he consented to an operation.

Physical examination negative; no skeletal abnormalities noted.

X-ray: "The auditory canal on the left is narrowed to about one-third of its normal size, probably throughout its entire length, but it is not occluded. The auditory canal on the right is almost completely occluded, but this shadow is not dense and the occlusion is probably very thin. Certainly this occlusion does not extend throughout the entire canal." (Dr. L. C. Kinney.)

Operation: Operation was advised and accepted. A modified meatomastoidectomy was done on both sides August 17, 1920. Wounds healed in two weeks, leaving a fair sized canal on both sides.

Hearing greatly improved subjectively. A. D.: Voice, 23 feet (full length of room); whisper, 23 ft.?; acoumeter, 23 ft.; C⁶⁴, plus; C⁴, plus; Rinne plus; Schwabach normal; Weber to left; Galton, full range. A. S.: Voice, 20 ft.?; whisper, 20 ft.?; acoumeter, 9 ft.; C⁶⁴, plus; C⁴, plus; Rinne plus; Schwabach slight increase; Galton full range.

The patient returned July 13, 1921, complaining of a return of the deafness and tinnitus in the left ear. Right ear still normal as far as he could tell. Fork tests showed a return of the left ear to about the original findings when first seen. Examination of the left canal showed the drum hidden by two deeply placed hard, insensitive, noninflammatory growths, sessile, one on the anterior inferior wall and the other on the posterior wall. A narrow slit only existed between them. The patient was willing to have another attempt made to relieve the deafness and the tinnitus. So August 10, 1921, complete meatomastoidectomy was done. A dry cavity remaining from the previous operation was opened and the entire posterior bony canal wall (i. e., what remained of it) was removed down to what was thought to be the annulus. A sessile bony growth 3x3x5 mm. was removed intact. Closure by means of Panse flaps. The small exostosis on the anterior inferior wall was not touched because of the feeling that enough had been done to relieve the symptoms. In about three weeks the entire wound was healed, the opening into the middle ear closing also. With the subsidence of all reaction the upper anterior portion of the drum was visible through a triangular opening about three mm. on each side. At this time it became apparent that the exostosis removed with the posterior wall lay external to the annulus which, as near as could be made out, had become enlarged, extending into and involving the drum. It had not extended inward so as to entirely occlude the tympanum, for medication applied to the canal could be discharged through the eustachian tube into the throat. At present the patient still complains of some tinnitus and very slight impairment of hearing, and the functional examination shows:

A. D.: Whisper 23 ft.; acoumeter 23 ft.; C⁶⁴ plus; C⁴ plus; Weber not lateralized; Schwabach normal; Galton full range. A. S.: Whisper 20 ft.; acoumeter 20 ft.; C⁶⁴ plus; C⁴ plus; Schwabach normal; Galton full range.

In the endeavor to discover the cause of this condition, especially since it seemed to be progressive, on the left side at least, an investigation of the patient's general condition was made. Stereoscopic plates of the sella showed "apparently nothing which gives definite radiographic evidence of a departure from the normal in this particular case." (W. O. Weiskotten, M. D.). The Wassermann reaction was negative. A study of the metabolism was made by Dr. Robert Pollock with the following results: The basal metabolism rate was -8 per cent. "Circulation, including heart, arteries and kidneys, fairly normal." "There is some slight evidence of arteriosclerosis beyond his years gleaned from the superficial arteries." "Blood pressure 140-90, 136-84; urine, specific gravity 1.013, light amber color, acid reaction, no albumen, sugar or acetone bodies, sediment negative; hemoglobin, 100 per cent; R. B. C., 5,200,000; W. B. C., 11,800. Differential count: P. M. N., 59 per cent; lymphs., 30 per cent; L. monos., 5 per cent; Trans., 4 per cent; Eos., 1 per cent; Baso., 1 per cent. No abnormal cells. The blood chemistry was as follows: Blood sugar, .09 per cent; sugar tolerance, normal; creatinin, 5 mgs. per 100 cc.; urea nitrogen, 25 mgs. per 100 cc.; uric acid, 3.3 mgs. per 100 cc.; alkali reserve, 43 cc. per 100 cc. X-ray of sella turcica showed a sella well within the bounds of normal." This shows a slight kidney retention of the creatinin, and also a slight reduction of the alkali reserve that could not be accounted for. Facilities were not at hand for a blood viscosity determination. An examination of the patient's family gave nothing startling in the line of evidence towards a family tendency, yet bears reporting. One daughter on the right showed what seemed to be slight thickening of the posterior canal wall; a second daughter, the isthmus on the right narrowed anteroposteriorly; a third, thickening posteriorly and deeply. A son also showed some anteroposterior thickening on the right side. An older brother of the patient had normal canals. Wife's canals normal.

The pathologic report of the specimen, by Drs. Grant and Wilson, is as follows: One portion showed "an osseous reticulum containing some slight amount of unchanged fibrous tissue. The osseous tissue itself contains a normal number of osteoblasts. In the few strands of fibrous tissue present, a

very small number of fibroblasts may be noted. A second portion consists essentially of fibrous tissue with considerable numbers of fibroblasts present. There is a moderate amount of calcification present arranged in minute areas and in minute lamellæ like structures. No osteoblasts or osteoclasts were found. This condition seems to be a proliferative periostitis." (Photomicrograph.) This growth can be called an exostosis just as any bony growth from a long bone. The process may be different in that (as has been shown) the tympanic plate is a "membranous" bone and not cartilaginous. According to MacCallum, "the membranous bones are produced by a change in the function of certain cells which acquire the power of laying down an intercellular matrix, in which they bury themselves, and which in time becomes calcified." The microscopic examination shows the various stages of such a process, from fibroblasts in fibrous tissue to osteoblasts in osseous tissue. This is to be expected, since the process had been progressing up to the time of the second operation. We have here, I should say, an idiopathic case with no evidence of local or constitutional irritation or basis of the new growth except the prolonged sea bathing.

Prehistorically considered, this subject is most fascinating. The literature naturally would be somewhat scanty, but I desire to make a resumé of what has been found before reporting the material of the Anthropological Department of the San Diego Museum. Blake, in 1880, examined part of the skulls in the Peabody Museum of Archeology and Ethnology in Cambridge. They were specimens from the stone graves of the mound builders of the Cumberland Valley, Tennessee. Of 195 skulls, 36 showed exostoses, 12 in both canals, 9 in the right and 15 in the left. Of the 54 total growths, 42 were on the posterior wall and 12 rounded, principally on the anterior wall. These skulls presented no evidence of syphilis. Ten of the cases came from one distinct locality and 6 from another, pointing to a racial if not a familial tendency. He also investigated 108 California crania, finding five possessing exostoses in one or both canals. He reported five cases out of six South American crania (Seligman), two out of nine Marquesas Island skulls, and one Fox Indian (Welcher), two from Peru (Turner), and six in 334 Peruvian crania almost

uniformly at the end of the canal and anterosuperiorly or posteroinferiorly (Wyman). A continuation of his research gave six exostoses in 37 crania, all from one mound in Arkansas, again suggesting a family tendency. Here, too, there was no evidence of syphilis, the long bones being examined for that condition also. Jackson found 25 cases among the 450 North and South American skulls in the Hunterian collection of the Royal College of Surgeons, the majority being from locations near the sea. He states that 25 per cent of the skulls from the prehistoric mounds of North America show this condition. Virchow found eighteen cases in 134 Peruvian skulls. In a personal communication Dr. Bruno Oettking of the Museum of the American Indian states that in their collection there are 88 skulls from San Miguel and 62 from the other Channel Islands. In the entire series two show exostoses of the aural canal. This gives a frequency of 23 per 1000 in the San Miguel crania, and 13 per 1000 for the whole series. "The exostoses are pea shaped, of solid texture and symmetrically situated at the anterior and posterior walls of the porus acusticus externus, thus corresponding to the endings of the originally incomplete annulus tympanicus. The porus is almost completely occluded; the pea shaped exostoses are flattened at their point of meeting without, however, producing coalescence." With Dr. Oettking's permission I wish also to give his conclusions derived from the Jesup material (North Pacific) in the American Museum of Natural History:

"Although exostoses occur at an increasing rate in the different modes (of deformation of the skull) while the undeformed have none, I have myself not come to a final conclusion as to a connection between deforming influences and the exostoses, since these happen also in undeformed skulls of other series. I should like, furthermore, to call your attention to the fact that no exostoses have been found in the immature (juveniles and infantiles), which is according to R. Virchow (*crania ethnica americana*), of some importance in judging the causality of the phenomenon." The figures are as follows: Three types of deformations were examined. The lowest figures were 2.3 per cent among males only, or 23 per 1000. The highest was 32.8 per cent among males and 15.4 per cent among the females of the same group, an average of 23.4, or

23 per 1000. In another type the females were twice as frequent in occurrence as the males, with a final average of 1.9 per cent.

The specimens to be reported are those of the Anthropological Department in the San Diego Museum. They were selected by Dr. Hrdlicka from the prehistoric cemeteries along the coast of Peru and mountains near the coast. In all, 4800 crania and parts of crania were examined in order to collect an exhibit of cranial pathology. Twenty-six deformed skulls showed five cases of exostoses in the canal and one in the middle ear just below the eustachian orifice. All of them showed more or less anteroposterior narrowing of the canals, especially on the left. Fifty-two crania chosen to show other lesions regardless of exostoses presented five occurrences. In one the external table of the mastoid had been destroyed by some pathologic process and a large cell uncovered. This contained a pea shaped rough exostosis about 2 mm. in diameter. Twenty-two temporal bones and 2 skulls were collected specifically to show this condition. All the various types described by the authors of our present day literature were found in these remains except those with a thin bony pedicle. These may have been present during the life of the individual, but the natural course of decay and maceration would have destroyed the attachments and thus the exostosis become lost. Only one specimen showed involvement of the floor, while four, in addition to growths on the tympanic plate, had small scale or barnacle like exostoses on the roof, a total of six separate excrescences. The total number of distinct exostoses was 84 from 46 temporal bones (including skulls) out of the 4800 crania examined. If we regard each temporal bone as coming from a different skull there was a total of 34 crania. Forty of the 78 growths limited to the tympanic plate extended to the annulus from the outer end of the canal, or over 50 per cent. In many others the canal was so occluded that definite information could not be obtained in this regard. They were equally distributed between the anterior and posterior walls, and all except seven were situated at the sutures, tympanomastoid or tympanosquamous. The growths not at the sutures were never single, but exostoses existed at those spots simultaneously. Four consisted of a diffuse thickening of the

canal wall. The number of growths varied from one per cranium to four per temporal bone. The majority were roughly oblong and extended the long way of the canal, taller at the outer end. The very small minority, in fact, only five, were round or pea shaped. Reduced to figures, there were: 17.5 distinct growths per 1000 skulls; 9.5 temporal bones involved per 1000 skulls; 7 skulls involved per 1000 skulls.

The only suggestion as to relative prevalence among the males and females may be derived from the 52 miscellaneous crania. Four males were affected and only one female. Considering the low percentage reported from the clinics and private practice of the present day, and postulating the premise that both in present life and in the selection of the exhibit an equal number of cases may not have been reported, we may conclude that this condition was more prevalent in prehistoric times. Contributors to the literature of this phase of the subject have given figures in support of this conclusion. Present day statistics, it may be noted, refer to the per cent of cases of aural disease, while those of prehistoric man are those of crania in general, irrespective of the aural condition.

The illustrations are photographs of some representative temporal bones selected to give a general idea of the exhibit.

As is well known, the long axis of the aural canal is not a straight line, the membranocartilaginous portion being directed inward and slightly upward and backward, and the osseous portion inward, slightly downward and forward. The junction of the two makes a rounded angle projecting into the lumen of the canal, forming a ridge from which each division descends. Immediately anterior to this angle is the mandibular joint (Politzer), and it is a well known fact that in mastication the movement of the lower jaw produces a change in the lumen of the canal, and that violence applied to the mandible at times causes fracture of the tympanic plate. Bruhl's explanation in otosclerosis that the stress due to the pull of the tensor tympani muscle accounts in many cases for the initial bone changes about the oval window, and Gray's belief that molecular stress on the bone in the same situation by movement of the stapes may in some way be productive of the otosclerotic process, seem both ingenious and plausible. In a similar manner, the writer believes irritation to the aural

canal due to mastication to be a contributory etiologic factor of aural exostoses. Evidence of this may be gathered from the investigation of the life and habits of early man. Antedating and even during the use of flint and obsidian tools, prehistoric man surely depended upon nature's equipment. While he no doubt was early in possession of deft hands, it seems only natural to assume that the teeth became a third hand, as it were, bringing the jaws into strenuous use, not only in masticating the tough uncooked foods, in cracking hard shell nuts, and assisting in preparing other necessities of life, but also as a weapon of defense. In most of the prehistoric crania examined the greater involvement is at the point of union of the cartilaginous with the osseous canals and along the suture lines. At the former point there would be not only the rounded eminence to receive special stimulation from any form of irritation within the canal itself, but the molecular stress exerted by such use of the mandible must have been considerable at this portion of the canal as well as along the unfused or weak tympanomastoid and tympanoquamous sutures, resulting in irritation of the more susceptible areas of the tympanic plate mentioned previously.

Referring once again to the literature on otosclerosis as being fruitful in its suggestions, I wish to quote from Gray's latest publication on the subject, taking the liberty of substituting the word "exostosis" for "otosclerosis." He says: "The other general aspect of the problem is that the majority of individuals never do suffer from exostoses, no matter how badly or from whatever cause their general health becomes affected, or whether they suffer from some local disease of the ear, such as suppuration, middle ear catarrh, etc., or not. Now it appears to the writer that, viewed from this point of view, the logical conclusion to be drawn is that the cause of exostoses—that is, the condition without which the disease cannot occur—is to be found in the organ of hearing itself, and, further, that this condition exists in the organ of hearing of certain individuals, and in these individuals only. From this point of view, which the present writer believes to be the correct one, exostosis is idiopathic. That is to say, the disease occurs in people who have an inborn tendency to it, and that this is the only constant factor in the etiology. No doubt dis-

turbances, either locally in the ears or constitutionally in the bodies of these individuals, may precipitate the onset of the disease, but no particular one of these disturbances need be present and in many cases they may all be absent. The single constant factor in the innate tendency to the disease and the fact that hereditary influences are evident in many cases is strongly in support of this view." This "constant factor" can be postulated, in my opinion, from a consideration of the evolution of the organ of hearing. It is a fact, well known by biologists (as mentioned by Gray in his book "Otosclerosis") that the first division of the hearing apparatus to develop was the static labyrinth, as shown by the fish. Later, as the animal kingdom became amphibious, the hearing labyrinth appeared. Still higher in the scale we find the bony canal developing up to its present adult form in the genus homo. Can we not answer our problem by using the term "atavism"? Here in this disease is an attempt on the part of the organism to return to its primitive state, an attempt to obliterate the hearing and the static mechanism in the reverse order of its development. This would be the natural order of retrogression, the latest developed being the first to suffer from the latent tendency or rather capacity of reversion that lurks in all forms of life, human or otherwise. The canals of our ancestors would be, in the light of this fact, less stable than those of our immediate predecessors or fellow citizens. This may be regarded as one of the factors accounting for its much greater prevalence in primitive man. Those cases attributed to purulent processes in the middle ear and those in which the condition arises apparently spontaneously can be reconciled on this common ground. Why do not all cases of O. M. P. C., with or without polypi, develop exostoses? The purulent process merely calls into action the latent tendency of the organism. The many other factors, rheumatism, syphilis, gout, etc., may act in the same manner. But underlying the whole process we can postulate the theory I have advanced. All the divergent clinical evidence that has been brought to light since the earliest discussion of the subject can thus be reconciled—i. e., the many etiologic factors, the absence of any such, the hereditary element and the sporadic appearance in an individual with no apparent hereditary taint.

SUMMARY AND CONCLUSIONS.

Exostoses are relatively a rare condition in the aural canals at the present time.

They may exist along with some irritative condition in the ear, inflammatory or noninflammatory. Constitutional disturbances have also been found to exist coincidentally. These may all be absent and the condition linked with a hereditary, congenital or developmental factor.

Practically all the exostoses found exist along the lines of union and of the tympanic plate and the rest of the temporal bone. It is in these two regions that the bony canal is developed most rapidly from the annulus. It may be postulated that these ends of the annulus that proliferated the more rapidly are more susceptible to irritation of any sort.

An additional factor in the development of exostoses along the suture lines at these more susceptible areas is the irritation due to mastication acting on what are in many instances weakly fused lines of union.

The case reported offers little light on the subject of the etiology except that excessive sea bathing may have been an exciting factor. The process microscopically and pathologically considered was a "proliferative periostitis." Accompanying the exostoses were an intumescent rhinitis, a markedly deviated septum and an O. M. C. C., conditions which, like those complicating an otosclerosis, may often be neglected or even overlooked. In all cases coexistent pathology in the head should be corrected. The general condition of the patient should be investigated at the time of examination and with regard to the circumstances at the onset of the aural symptoms. In this way many interesting and relevant facts may be uncovered.

Prehistorically considered, aural exostoses were more prevalent in primitive man. The etiologic factor postulated in this regard is the excessive use of the teeth, reacting on the more susceptible areas before mentioned (still more susceptible because of the earlier stage in evolution) producing lines of increased stress and even slight motion at the suture lines, very often weakly fused or largely unfused.

In view of the many causes given, we can conclude that the fundamental factor has not been discovered. I wish to advance the theory of atavism as a possible answer.

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VIII.

CONSIDERATION OF THE RADICAL MASTOID.

By C. M. SAUTTER, M. D.,

NEW YORK.

The object in reporting radical mastoid cases is again to emphasize the seriousness of chronic suppuration and to impress the full responsibility that otologists bear in the education of the laity. It is not the writer's intention to dwell on the local treatment of chronic suppuration. In reporting the features of fifty cases, it is at once obvious that there was no choice between treatment and operation. The radical mastoid operation has been greatly stigmatized by otologists, but when one considers the early technic employed in radical surgery it is easy to understand why it was so universally condemned.

In choosing this subject, the writer has been prompted partially by the increasing interest in the radical mastoid manifested by students, especially those who have practiced otolaryngology for some years. It is possible to stretch the imagination to see the day when this operation may be more universally done and with very little anxiety on the part of the operator. Untoward accidents may happen at times, but after experience on many mastoids, both on the cadaver and the living, one must progress in his technic or he will consistently fail.

The compulsory examinations of the late war, during the draft, brought to notice many cases that would otherwise have escaped observation. Every other ear case brought to the attention of the Advisory Board was one of chronic suppuration. Since the close of the war, the writer has been active in the U. S. Public Health Service and has been surprised at the large number that require operation before a cure can be effected. A large percentage of these cases applying today are due to the epidemic in the cantonments and to trauma produced by explosion of shells and concussions at the front.

Nature itself has pointed the way to treatment of these cases. One now under observation illustrates this most forcibly. In this chronic otorrhea of cholesteatomatous character had performed a complete radical operation, with the exception of enlarging the concha. The cavity proper is as pretty and clean as any performed by human hands.

The indications for operation may be briefly considered, subjectively and objectively. There may be a history of headaches confined to the involved side, vertigo, foul discharge, tinnitus, defective hearing and impaired general health. Otoscopic examination may reveal foul discharge, a mastoid fistula, granulations, cholesteatoma, the presence of sequestra, exostosis, or necrosis about the tympanic cavity. Any one of these symptoms, if manifested in sufficient intensity, may warrant operative interference.

COMPLICATIONS.

The complications demanding surgical interference are acute exacerbations, meningeal infections, labyrinthitis, sinus thrombosis, facial paralysis, retropharyngeal and deep cervical abscesses.

Essentials of Surgical Technic: Much of the success of the radical operation depends on the mental condition of the surgeon at the time of operation. He must be able to think clearly and concentrate his mental efforts upon the physical requirements. Good tactile sense and visual acuity are necessary attributes, of course, and good judgment must be exercised.

Instruments: On the whole, the earlier instruments, the gouge and curette, have proved to be the most practical, although some prefer to use an electric burr. By exercising perfect control of the gouge and directing upward all motions of the curette, it is found that the instrument for the protection of the facial nerve is unnecessary when removing the facial bridge. A good reflecting light is indispensable in doing the radical operation.

Anesthetization: In administering the anesthetic, keen watchfulness on the part of the anesthetist is important for the successful termination of the operation. Close observation of the face on the involved side should inform the surgeon of

any trespassing on the facial nerve. In pulmonary conditions ether should not be used; nitrous oxid is the anesthetic of choice.

SALIENT POINTS TO INSURE SUCCESS IN THE RADICAL OPERATION.

1. The dural plate, whether over the tympanum or the antral area, should be removed if necrosis is suspected. Whenever cholesteatoma is present these plates are always involved and necessitate removal.

2. After removing the bridge, a slight amount of "overhang" of the facial ridge, just above the stapedial area, is allowed to remain until near the completion of the operation in order more carefully to protect the stapes.

3. In curetting or sponging the tympanic cavity, all movement should be directed from the floor to the vault or vice versa. In directing the movement in this manner there will be little danger of fracturing or dislocating the stapes, which may introduce infection into the labyrinth, or ultimately cause impairment of hearing.

4. In curettage of the eustachian tube, greater force should be directed forward and above and less posteriorly, because of the near proximity of the carotid artery. A reverse or ring curette is the instrument of choice here.

5. The anterior posterior diameter of the tympanum should be sufficiently enlarged to allow ample room for ventilation and drainage. This may be done by removing the overlying edge of the internal surface of the posterior canal wall, and reducing the convexity of the bone of the glenoid fossa in front.

6. The cells about the processus cochleariformis and those overlying the eustachian tube should be thoroughly exenterated.

7. The inferior wall of the tympanum or hypotympanum should be reduced so as to prevent the formation of a pocket. It should be especially lowered when the disease of the mastoid extends back to the tip.

8. An adequate flap of the concha is necessary for post-operative treatment, in order to permit ventilation and tone vibrations to be more readily conveyed to the internal ear. From a cosmetic standpoint, the flap should be broad at the base and more narrow at the top, although in women the ear

is later covered by an abundance of hair. Where the cavity of the mastoid extends well back into the infrasinus area or tip region, a fairly large flap is preferable for the ready inspection of the cavity, although a small pharyngeal mirror may be used to aid in this.

SURVEY OF CASES AND COMMENT.

Bilateral suppuration, as shown by these cases, assumes a fair proportion. Ten of the fifty had a double otorrhea. On four of them a double radical operation was performed. Two cases which were skin-grafted were able to hear a moderate whisper at from sixteen to eighteen feet. One of these had previously been a student at a deaf institute. The mother of this patient returned to the hospital for a certificate to allow her boy to enter the public school. A third patient improved so much in general health and hearing that the other ear was operated on; the result, however, was not so good with the second ear, partly on account of some necrosis coming from the dural plate, which was overlooked. This patient was one of the earliest cases. A secondary operation has been advised in order to eliminate completely every vestige of disease, as the hearing does improve when the ear is dry and aided mechanically by the use of a plug. The fourth of these bilateral radical cases was a former service boy, operated upon at one of the U. S. Public Health hospitals. The improvement here in the first radical was so marked that he insisted on having the other ear operated also. Three of the series had previously had simple mastoidectomies. In one case this had been the operation of choice as a conservative measure; and the continued suppuration following this necessitated a radical operation. The other two patients had very foul discharge and a high degree of impairment of hearing. The canal walls in both instances had collapsed so that no view of the tympanum could be obtained. In other cases it has been observed that whenever a simple mastoid has been performed and the canal wall allowed to collapse the insufficient ventilation of the tympanic cavity results in chronic necrosis of this cavity. In both instances after the radical, pus was excessive, so that the posterior wound broke down, forming a permanent fistula, the cicatrices from the former

operations with insufficient suturing probably accounting for this. The lesson to be learned from this is that the periosteum should always be sutured in addition to the closure of the skin. The ears later became perfectly dry, and the audition of the patients was much improved.

In another case, a patient thirteen years old had had a discharge since two years of age. She had been under persistent local treatment for the last two years, with no improvement. Her hearing was very poor. A radical operation was performed seven weeks ago. No skin graft was employed because of the profuse discharge at the time. This case well illustrates, however, how ineffective local treatment may be in some instances if one does not understand the subject. One month after the radical operation, during the absence of the writer, the ear was again examined and found to be filled with granulations and a profuse foul discharge. This could have been avoided had the patient been treated with proper diligence and after care. After six weeks of persistent careful treatment a dry cavity was secured. With the noise apparatus in the other ear this girl was able to hear moderate voice at twelve feet. The general health of the patient improved so that there was a gain of nine pounds in the last month.

In this series the youngest patient operated upon was three and a half years of age; and the oldest, fifty-five. With the child, there was some hesitation about doing a radical operation, but the cavity was found to be filled with cholesteatoma with extensive necrosis, so that it was imperative. The case was skin grafted and resulted in complete cure except for an occasional tubal discharge coming on with an acute rhinitis.

Recently the writer performed a radical mastoid operation on a child of two years. This patient appeared to have a subperiosteal abscess with a complete congenital canal atresia. The mastoid and middle ear were filled with pus and cholesteatoma necessitating a radical.

Recently two cases have been observed in children slightly over two years of age, both of them requiring a radical mastoid operation. One of these was admitted to the hospital with meningitis, due to the extension of mastoid

necrosis. Cholesteatoma was present in both cases; this form of infection evidently comes with the inception of the primary otitis, and persists until it is either overcome by the surgeon or it overcomes the patient.

One case of the series was complicated by pulmonary tuberculosis, which was not recognized until the administration of ether, which provoked a persistent cough. The pulmonary condition was no doubt aggravated by the ether. This patient died in a sanitarium four months later. In clinical cases one is apt to attribute depleting symptoms to otorrhea. Whenever possible an examination should be made to rule out any complicating diseases.

In all the writer's experience, syphilis was suspected in only one case of mastoiditis. This occurred in an acute infection. The distinctly gelatinous character of the mastoid bone in this instance prompted a Wassermann test, which proved to be four plus. The resolution in this case, however, took place in the usual time, and later the patient was referred for syphilitic treatment. In none of the series was there evidence of syphilis, although a positive infection does not necessarily contraindicate an operation.

Two cases of the series of fifty were complicated by epidural abscesses. In both instances extensive pathologic changes had taken place for some time, without any special discomfort to the patient. The first of these illustrates how dangerously near one can approach a fatal termination by adopting a watchful waiting policy. The writer was well acquainted with this patient and tried his utmost by local treatment to avoid any surgical interference. This man was forty-six years of age and had had a discharge ever since he could remember. The discharge increased, with an acute attack of rhinitis. This was followed by a sensation of heaviness about the same side of the head. No temperature or tenderness were present, but after two weeks of daily observation, the head symptoms persisted, with three nights of insomnia. On the morning following the third night of sleeplessness, the patient complained of an acute pain behind the eye on the side of the involved ear. A diagnosis of epidural abscess was made because of the coincidence of the eye symptoms, and the patient was operated upon that same afternoon.

The destruction of bone in the mastoid was almost unbelievable. The sinus wall was completely necrosed. When attempting to pass the probe into the aditus to find the antrum it was necessary to slightly displace the projecting and exposed lateral sinus and while doing this twice a couple of drams of cerebrospinal fluid escaped. The spinal fluid apparently came from the region of the internal auditory meatus. The sinus was closely approximated with the exposed dura. The tympanum was filled with debris and granulations. In removing some of the granulations and edges of the diseased bone about the dura, a teaspoonful of pus escaped. The facial nerve was exposed by disease and twitching was observed during the operation when sponging the tympanic cavity. A spinal puncture was done immediately following the operation. Examination revealed a cell count of 80 per cubic mm. No complications developed. Undoubtedly the high spinal cell count was due to the intimacy of the abscess with the dural sheaths, resulting in a protective form of meningitis. The ear subsequently became perfectly dry and hearing was much improved.

The other case occurred in a boy of seven years, who was unable to describe his symptoms accurately. During the day he had been at play as usual, but at night he complained of pain about the head. A chronic suppuration from which the child had suffered, made the parents suspicious. A radical operation was performed by the usual technic. The cavity was apparently one of those of a constricted type that so often follow long suppuration, where there is sclerosis and proliferation of bone. Only the habitual practice of following up the smallest bit of granulated tissue led the operator to discover the epidural abscess cavity. In this instance the spinal cell count was normal. The patient refused a subsequent skin graft, which was advised several weeks later. There is still an odorless serous discharge from the tubal area and the region where the epidural abscess had formed has failed to granulate. The boy's hearing, however, has considerably improved.

The coincidence of facial paralysis with chronic suppuration must be considered as a positive symptom of bone necrosis, although a facial palsy may be the result of other conditions.

When the facial nerve is exposed by disease, it is usually evident along the internal surface of the posterior wall or where it comes to the surface as it crosses the tympanic cavity. In two cases of this series there were large sequestra, one involving the cochlea, semicircular canals and petrous portion of the temporal bone. In this case there was no paralysis prior to the operation, but in removing the sequestra the facial nerve was unfortunately destroyed. In the second case the facial nerve was exposed through the entire route of the tympanic portion of the posterior wall with no palsy and none following the operation. In the after treatment the facial nerve was plainly seen in its course. Cases with facial twitch occurring during the operation are usually followed by temporary paralysis a couple of days later.

In the last two cases in which the writer has exposed the nerve during the operation he has removed the packing after twenty-four hours and replaced it by a light dressing, instead of waiting five days, and to this early removal he ascribes his success in preventing a temporary facial paralysis. In three other cases where the packing was allowed to remain for five days the purulent inflammation produced a perineuritis with a facial palsy, so that the patients were unable to regain the function of the nerve until after five to ten weeks. In none of these cases, however, was there any permanent paralysis.

In the practice of the writer, three patients have been seen with partial ankylosis of the inferior maxillary following a radical mastoid. Two of these were operated upon by other surgeons; one was kept under observation for a little more than a year, when the ankylosis disappeared without further intervention. The last patient was entirely free of the impairment in three weeks. It has been observed that this complication follows a fracture or slight exposure of this articulation when trying to reduce the convexity of the anterior wall of the canal. Should this accident happen it is far better to purposely expose a fair sized area of the glenoid fossa so as to prevent synovitis and ankylosis.

Another patient was admitted to the hospital with a four day history of acute exacerbation with sinus thrombosis. There were three chills, lasting ten minutes each. The temperature range was from 103 to 106 deg. F. Upon opening

the cavity, the dural sinus plates were completely broken down. A large clot was visible in the lateral sinus. The pressure exerted by this clot was sufficient to cause a depression in the cerebellum about the size of a walnut. The patient's pulse rate, which was from 50 to 60 per minute, caused a suspicion of some cerebral lesion. In this instance it is believed that simply the pressure of the brain tissue was sufficient to cause the interference with the circulation, resulting in the very low pulse rate. Because of the serious physical condition at this time, only a resection and ligation of the jugular was done with curettage of the lateral sinus,—the radical mastoid being deferred until a month later. The foul discharge had continued from the diseased tympanic cavity. A skin graft was applied, and the cavity became perfectly dry with improved hearing.

In two other cases of the series the lateral sinus was very large, exposed by disease, and lay against the posterior wall so that it was impossible to remove the facial bridge except by working from the tympanic side.

In two more cases in which the radical mastoid was performed the lateral sinus wall was found to be so thin that at the time of the removal of the first dressing five days subsequent to the operation there was a profuse hemorrhage. In the one instance the hemorrhage was repeated during several weeks upon succeeding efforts to remove the gauze. Both cases ran a high temperature for several days, typical of sinus thrombosis. A blood culture was made with negative results. Later the temperature fell to normal.

These four cases furnish a warning to those who advocate treatment by suction for the cure of chronic suppuration. A thin or exposed sinus wall may easily be infected or injured in this manner.

SUMMARY.

Reviewing this series of fifty cases: there were twenty-three operated upon with a primary skin graft. Six of the total number still have a discharge from the eustachian tube, four of them children. Only one case of the series has evidence of a necrosed dural plate, which was mentioned in one of the preceding paragraphs.

COMPLICATIONS EXISTING PRIOR TO RADICAL MASTOID.

Epidural abscess	2
Sinus thrombosis	1
Sequestration	2
Facial nerve exposure.....	3
Secondary mastoiditis	3
Labyrinthitis (suppurative)	1
Acute exacerbation	12

POST-OPERATIVE COMPLICATIONS

Erysipelas	2
Pneumonia	1
Temporary facial paralysis (duration three to eight weeks)	4
Exacerbation of pulmonary tuberculosis.....	1

In this connection several points should be emphasized. To begin with there was a large percentage of cases in which there was an acute exacerbation, necessitating the radical operation. In such cases a primary radical may be safely performed if the patient's vitality permits. Only three died and in none of these was death directly attributed to the operation. One of the three cases was complicated by pneumonia. A second patient came to the hospital with symptoms of labyrinthitis and died several days later of meningitis. The third case died of pulmonary tuberculosis four months after the operation.

In a large number the radical operation was imperative because of the complications. The risk involved was very slight. In none of the fatalities was death directly due to the operation. In only one of the entire number did the discharge from a necrosed area continue after operation, and this was one of the early cases of the series, due to the failure to remove the diseased dural plate. In twenty-seven of the fifty cases the dura was either partially or almost completely exposed either purposely or by disease. This large percentage of dural exposures doubtless accounts for the dry cavities obtained.

In most instances the hearing improved, the exceptions being a few that had unusual hearing before operation. The contention has been made by a number of otologists that the

improvement is only temporary. The writer maintains that by careful after treatment, immediately following the operation, keeping down the granulations and preventing adhesions from forming the surgeon can do much to prevent subsequent impairment of hearing. Later treatment also is important. It consists in removing all ceruminous and desquamating epithelial material as it forms, followed by the application of albolene. The local application of the plugs or emollients restores the hearing, if temporarily diminished.

The general health of the patient is invariably improved, as manifested by an increase of weight and the return of a healthy color to the skin.

CONCLUSIONS.

1. The risk to life is almost negligible.
2. Age is not a factor in considering a radical operation. The indication depends on the symptoms and the condition of the patient.
3. When the operation is properly performed, the hearing is improved unless there is some disease of the receptive mechanism. A radical should be advised where the hearing is very much impaired to improve the audition.
4. The operation is rarely followed by permanent facial paralysis.
5. The application of skin grafts increases the percentage of cures, materially lessens the time of resolution and aids in the preservation of hearing.

11 EAST 48TH STREET.

IX.

CASE OF POLYARTHRITIS COMPLICATING MASTOIDITIS. OPERATION. RECOVERY.

BY DUNBAR ROY, A. B., M. D.,

ATLANTA.

Focal infections as a cause of arthritis seem now to be an established fact. However, mastoiditis, whether acute or chronic, seems to have received but little mention as one of these causes if we may judge from the infrequency of reported cases.

While various conditions such as glycosuria, convulsions, and other nervous phenomena often occur in the course of acute infections of the ear and while there seems to be no good reason why rheumatism, arthritis and neuritis should not occur from acute and chronic ear affections, yet the complications which do occur are the conditions which are usually denominated as pyemic.

In looking over my own personal histories of aural diseases for the last twenty-five years the writer has been unable to find any other case even resembling the one here reported and the literature examined would certainly lead one to the conclusion that such a complication is exceedingly rare. What few references as have been found have been reported in this country so that one is forced to the conclusion that such complications are so frequent as not to be worthy of mention or that the same has not been observed.

Case history: Jack J., white, age 4. Strong, healthy looking child, giving only the history of repeated colds in the head.

Examination shows tonsils very much enlarged and also the presence of increased amount of adenoid tissue. Operation advised and on February 2, 1921, at Georgia Baptist Hospital both tonsils and adenoids were thoroughly removed by my associate, Dr. Murdock Equen. Convalescence was uneventful and in a few days the child was out and immediately began to improve.

On March 5, 1921, nearly a month after the tonsil and adenoid operation, the patient began to complain of severe pain in right ear and two days later the left ear also began to pain.

Dr. Equen was called in consultation and finding both ear drums red and bulging, under chloroform anesthesia, these were freely incised. This was followed by a free discharge of mucopus from both middle ears, all pain being relieved and the temperature being practically normal. Both ears were kept irrigated with a hot carbolized solution. The patient seemed apparently well. Appetite good and sleep uninterrupted. There was no pain, tenderness or edema over the mastoid. With the exception of the profuse discharge there were no other complicating symptoms.

Smears taken from the discharge showed the presence of both staphylococci and streptococci organisms. To all appearances the case was progressing nicely and healing of the middle ear seemed to be taking a normal course. The only exception to this was the continuous free discharge of mucopus.

On March 9th, four days after the beginning of the middle ear discharge, the patient was seized with an acute arthritis of the finger, knee and ankle joints on both sides. This was accompanied by a sudden rise in temperature to 102 F. with intense pain on moving these joints and pain even without movements. Patient was unable to sleep day or night without anodyne. The usual remedies for this condition were tried without success and after a consultation we decided that there was a toxic absorption from the middle ear indicated by the very profuse discharge and while there were no typical mastoid symptoms it was evident that this profuse discharge could only be coming from the mastoid antrum and cells. The urinary examination showed nothing abnormal. No blood count was made as we did not deem it necessary.

Under the circumstances we decided to open the mastoid. Accordingly on March 12th, one week after the membrana tympani was punctured, the right mastoid was operated upon, this being the first ear involved and from which the discharge was most profuse.

A simple mastoidectomy was performed. On entering the mastoid antrum pus was found and a few adjoining cells in-

volved. These were rapidly cleaned out and a free communication made with the middle ear. The wound was closed entirely and a small wick of iodoform gauze placed at the lower end of the wound.

In one week's time all discharge had ceased and the mastoid wound was entirely healed. There was only a slight amelioration in the joint symptoms and the discharge from the left ear was now very profuse. Consequently one week after the first operation the left mastoid was opened and almost identical findings resulted. The wound was closed in the same manner. It took only twenty minutes for each operation.

For a few days there was considerable discharge from the external canal and also from the lower part of the wound. However, this rapidly disappeared under treatment and in less than two weeks the mastoid wound was entirely healed and the middle ear was dry. The hearing in both ears was normal. Two days after the operation all arthritic symptoms in the joints disappeared and the patient has had no further trouble. In addition to the double mastoidectomy autogenous vaccins were made from the middle ear discharge and five injections were given in increasing strengths.

As has been mentioned previously, it is surprising how few cases of a similar character have been reported. While my tabulated report is perhaps by no means complete the writer has appended an abstract of all the cases which he could find and which will at least aid in further research along this line.

Purulent Pheumococcic Arthritis in Children. Dudgeon and Brausen, *Lancet*. 2:316, 1903: Five cases are reported, only one of which is related to otitis.

Case 11.—A boy, 6 years, admitted March 23, 1903. The patient had been ill for three weeks with earache and subsequent otorrhea, when on March 16 he complained of swelling in the left knee joint, followed on the next day by similar affections of the right hip and wrist. There was no history of rigor, convulsion, or any other symptom of note. The patient on admission appeared to be very ill and lay curled up in bed with all his joints flexed. He had a troublesome dry cough and was slightly delirious, though his fever was moderate.

Nothing abnormal was to be discovered in the lungs or heart. The left knee joint was swollen, red, hot, shiny and fluctuating, while the right hip and wrist appeared to be in a less advanced state of inflammation, and this in the latter seemed not to involve the joint, but to lie superficial to it. Urine was normal. The knee joint was opened and a quantity of thin pus and flakes of fibrin were evacuated and a similar result attended the opening of the hip joint. Little benefit resulted from the operation though the wounds progressed favorably. Fever continued high at night with morning remissions and with gradual loss of strength, but without specific complications; the child died from asthenia twenty days after operation. No post mortem was allowed.

In this series of five cases, the exciting cause of the arthritis in three was found to be "pneumonia," "bronchitis" and "otorrhea."

A table of Netter, quoted by Muir and Ritchie, gives otitis media as a source of infection (arthritis) in children in 29 out of 46 cases collected, bronchopneumonia in 12, meningitis in two, pneumonia in one, pericarditis in one, and pleurisy in one. No case of primary arthritis occurs in the list.

Mastoid Cases at Camp Sherman, Christian R. Holmes. *Ann. Otol., Rhinol. and Laryngol.* 28:1, 1919.

In a series of forty cases reported of mastoid diseases with complications one is given as double.

Case 34.—Evans, double otitis media. Simple mastoidectomy left side, March 14. Temperature 106.5 (rectal) at operation. Fell to normal and remained so for about a week, when inflammation developed in left knee, with moderate rise of temperature. Capt. Sturgis aspirated. Contained mucopurulent fluid. April 3 it became necessary to make a simple mastoid operation on the other ear, at which time the knee was aspirated again. Fluid dark and cloudy.

Infective Arthritis Complicating Otitis Media. W. P. Eagleton, *Arch. Otol.* 34:483, 1905.

Case 11.—Male, aged 24. Previous history negative. Never suffered from rheumatism. Otitis media suppurative following coryza. Paracentesis made under nitrous oxide.

Very profuse discharge. Temperature 101-103 degrees F. One week later pain in right knee, accompanied by only slight swelling. The next day, the left knee was similarly affected, and later both ankles, accompanying which a distinct pericardial or possibly an endocardial murmur could be heard, which was not present on the previous day. Gradual recovery.

In true infective arthritis and that complicating osteomyelitis, the mortality is very high, while the mobility of the joint is almost sure to be seriously impaired, if not altogether lost. The author found only 79 cases reported pneumococcic arthritis, of which 60 per cent (48) died.

Case of Mastoiditis Complicated by Suppurative Arthritis of the Shoulder. Erskine. *Ann. Otol., Rhinol. and Laryngol.* 25:1035, 1916.

The mastoiditis developed after an attack of influenza. The patient had been confined to bed for a week and then after having had an earache for a day and a half the ear was examined and a hemorrhagic bulla was found on the drum membrane. The drum was incised and drained. The patient improved for five days, then again suffered with earache. After a week, a culture showed streptococcus brevis. A mastoid operation was performed about which there was nothing unusual. The man was over six feet tall, below par, weighed 147 pounds.

On coming out of the anesthetic the man complained of pain in the shoulder of the opposite side; he declared that it felt as if it were dislocated. There was tenderness over the front of the shoulder and the movements were painful. The next day it showed redness and three days later, when the joint was opened, the arm was very much swollen. The temperature was 102 to 103 degrees F. The man had profuse perspiration but no chills and no drop in temperature. The blood culture was negative. A general surgeon opened the joint and found three ounces of serous pus. The culture from the mastoid showed streptococcus, the culture from the pus of the shoulder showed streptococcus, as did the pus from the ear before the mastoid. Immediately after the incision of the shoulder joint the temperature went down. Subsequent course uneventful to recovery.

Dr. Opdyke told of the case of a boy with a mastoid in the right ear and arthritis in the elbow, knee and ankle joint. A year later he had a mastoid on the other side and the same involvement of the other ankle, knee and opposite elbow. The ankle, knee and elbow were not opened in either instance, though they were involved for a long time.

A Case of Pneumococcic Pyemia with Recovery. A. T. Davies and W. L. Brown; *Lancet* 2:1017-1020; London, October 8, 1904.

1. A table is given of thirty-nine fatal cases of multiple pneumococcal infection occurring at St. Bartholomew's Hospital, London, between January, 1894, and August, 1904, where the lesions were bacteriologically proved to be due to the pneumococcus alone, the probable course of events is indicated and the pathologic report appended in each case.

2. Case 35 listed girl 13 years old, shows the primary exciting cause as otitis media; complications arising are infective mitral endocarditis, rupture of tendinous cords, splenic and renal infarcts, suppurative arthritis of the right shoulder—and wrist joints; pneumococcus bacillus was cultivated from vegetations; no detailed history is given nor any nearer date than that the case came up "in the last ten years."

Relation of Infective Foci to Rheumatoid Arthritis. J. Lindsay; F. S. P. Strangenays, ed. *Bull. Com. f. the study of Spec. Dis.* 2, No. 3:106-116, Cambridge, Engl., 1908.

1. Report is based on 172 cases of rheumatoid arthritis at Royal Mineral Water Hospital, Bath, Engl.

2. The 172 cases included 138 females, 34 males; information on definite source of infection preceding onset of disease obtained in 73 females, 15 males.

3. Otorrhea present in 7 out of 138 females, in 3 out of 33 males.

Case Reports on Otorrhea. Case 48.—Married, printer, aged 43; onset of rheumatoid arthritis ten years ago; complicated by spondylitis deformans; has had discharge from left ear for many years.

Case 55.—Married woman; age 57; onset four years ago; prior to this abscess in left ear from picking ear with hairpin; intermittent ear discharge since.

Case 101.—Married woman; age 31; double otorrhea; very offensive discharge present in both ears from childhood.

Case 104.—Girl; age 12; discharge from both ears since childhood; onset at age of 7, slight discharge from left ear on admission to hospital.

Case 108.—Boy; age 11; onset 12 months ago; intermittent discharge from right ear since age of 6 or 7; discharge recurs apparently on exposure to chills.

Case Histories of Pneumococcal Osteomyelitis. Blecher; Deut. Ztschr. f. Chir. 48, no. 4:413-417; Leipzig, June 30, 1898.

Case history: 1. Boy; age 12; spontaneous left otitis media purulenta beginning November 2, 1897, subsiding two days later; November 5 painful swelling in right femur; rigor and general malaise; admitted to surgical clinic November 8.

2. Clinical findings: Well developed boy; temperature 39.9 degrees; pulse 110; skin over right femur reddened; knee much swollen; lower third of femur painful to touch; deep effusion into knee joint; left ear dry; defect of drum membrane; tympanic membrane moist and shiny.

3. Operative findings: Medial and lateral incisions made on femur; two isolated abscesses found below detached femoral periosteum, both communicate with the upper recess; bilateral arthrotomy of knee joint; drainage of pus; iodine gauze plug; dry bandage.

4. Microscopic findings: Pure cultures of Fraenkel-Weichselbaum's capsulated diplococcus were obtained both with pus drained from wound and with secretion from left ear; secretion from nose contained the same bacillus, but no pure culture could be obtained; otitis media was therefore primary cause of arthritis.

5. Postoperative course: Fever persisted until end of November though no new focus of infection or any retaining of pus could be demonstrated; pus formation decreased gradually; in December temperature became normal; granulations formed and on January 15, 1898, the wounds were well healed; after recovery there was still marked restricted motion of the knee joint and a moderately painful swelling of the lower femoral end.

Acute Otitis Media as an Early Symptom in Acute Rheumatoid Polyarthritis. C. Wolf; Arch. f. Ohrenhkl.; 41, Nos. 3 and 4; 213-216; Leipz. Dec. 30, 1896.

Case of Bilateral Otitis Media and Acute Polyarthritis. Male, 50 years old; left subacute otitis media and diminished hearing June 8, 1888; three days later slimy, flaky secretion, hearing improved, on day following right otitis media; drum membrane incised and bloody, serous, later slimy, flaky secretion drained; pain relieved; evening of fifth day rise in temperature to 38.6 degrees C; temperature on sixth day 30.2 degrees C; headache, pain in limbs, loss of appetite and general strength; on seventh day symptoms of acute rheumatism, painful swelling of both metatarsal joints; the disease progressed, many other joints becoming involved in succession; endocarditis was an added complication; by the fourth week both tympanic cavities healed but still showed slight thickening of the epithelial layers; hearing diminished to about half of normal acuteness; one and half years later death occurred from endocarditis.

Case of Right Otitis Media and Inflammatory Polyarthritis. Male, aged 18; slight angina April 1, 1896, four days later right otitis media; livid, moist, bulging drum membrane; hearing almost gone; bone conduction to right increased; rinne positive; temperature 38.6 degrees C; April 5, incision of drum membrane, draining bloody, serous secretion; April 9 no pain, no secretion, improved hearing; on eighth day incision healed, slight temperature, general health unimproved, continued constipation, anorexia, sudden rise in evening temperature 39.5 degrees C, severe pain in various body portions, particularly in neck and back; no proof of any other diseased organ established; on ninth day marked swelling of right knee joint; successively most other joints became involved, finally the cervical vertebral articulation also; in the course of inflammatory polyarthritis considerable pericardial exudate developed; later exudation developed in right pleural cavity; several attacks of cardiac weakness; irregular pulse up to 124 per minute; emaciation; gradual improvement after ninth week; complete recovery after a prolonged stay in the country; re-examination October 23, 1896; tendinous thickening of right drum membrane, handle of malleus and short process of the

malleus more prominent than in left ear, hearing in right ear 6 m. for whispered speech, in right ear 12 m.; November 6, 1896, recurrence of inflammatory polyarthritis in right shoulder joint and exudate in right pleural cavity, no preceding angina or otitis media; recovery on tenth day.

A Case of Acute Articular Rheumatism Complicating Acute Otitis Media. Joseph Popper, *Med. Rec.* 99, 270, Feb. 12, 1921.

Report of a case of acute articular rheumatism following acute purulent otitis media and considered interesting, both on account of its rarity and because of the fact that this condition may be so easily confused with the bacteremic condition following severe cases of middle ear disease usually complicated by mastoiditis or lateral sinus thrombosis.

The patient, male, nine years old, was admitted to the hospital complaining of discharge from the left ear and pain in the right ankle. The child had had all the usual diseases of childhood with good recovery. His tonsils and adenoids had been removed two years ago. The family history was irrelevant. Ten days before admission, he had been put to bed with a temperature of 102 degrees F., complaining of headache, running nose and slight, dry cough. Two days later pain developed in the left ear, from which there was a spontaneous discharge of pus that night. The fever ranged between 101 degrees and 104 degrees during the ten days prior to admission. The discharge from the ear became scanty, meanwhile, and on the eighth day of the illness, an otologist enlarged the opening in the eardrum without, however, increase of the discharge following. On the ninth day there was pain in the back, and on the tenth day pain in the right ankle. Physical examination showed that the head, eyes, right ear, mastoid, neck, heart, lungs and abdomen were normal. Spine and reflexes were normal, as were the urine and blood. There was a small amount of mucopus present in the left ear, but the drum was not bulging. Following admission, the patient experienced pain, tenderness and disability in almost all the large joints, and some of the small joints of the hands. By the sixteenth day the temperature, which had been ranging from 101 degrees in the morning to 104 degrees in the after-

noon, declined and reached normal on the nineteenth day. With this decline, the symptoms cleared up and the general condition improved remarkably. The patient had been treated expectantly until the blood cultures were reported negative. This fact, together with the absence of chills, led the author to believe the case one of acute articular rheumatism, as seen in childhood. Salicylates were given and in twenty-four hours symptoms were markedly relieved. The author suggests that acute articular rheumatism may be a symptom complex secondary to some focus of infection, rather than a definite pathologic entity.

X.

THE END RESULTS OF RADICAL OPERATIONS ON THE ACCESSORY SINUSES.*

BY ROSS HALL SKILLERN,

PHILADELPHIA.

I don't know how often the question has been put to me, "Skillern, now as a man to man what is the ultimate condition of the patient after a radical on the frontal ethmoid?" or whatever sinus may be under discussion. As the frequency of this interrogation bespeaks its importance, I purpose with your indulgence, to base this dissertation upon the answer at least as far as my own personal experience is concerned. First of all, what is a radical operation on a sinus? I take it that when one speaks of a radical operation, one means an operation performed with the single thought in mind of an absolute relief from the symptoms with a more or less perfect cure regardless of the severity or extent of the surgical procedure. The last sentence expresses that which not infrequently takes place in that operation is oftentimes performed regardless of its extent or severity. That this expression has been repeated thus overly placing emphasis upon it is intentional and deservedly so, as did these not obtain, the question as to the ultimate results would never have been trite, but theoretical, and only of academic interest instead of representing the oft too trying reality which not infrequently arises in after months to plague those of us who are called upon to operate upon these cavities accessory to the nasal passages. When one considers the degrees in the severity of infection as well as the pathologic changes which have occurred, it is an extremely difficult task to approach this subject without entering into the details and finer minutiae. For the sake of clarity let us waive aside all so-called errors of judgment as to the advisability of the operation, overenthusiasm

*Vice-President's Address American Academy of Ophthalmology and Otolaryngology, Minneapolis, September 20, 1922.

of the operator or last resort methods and assume that a radical operation was clearly indicated, but despite its timely application, the end results were not everything to be desired. What do we mean when we say the end results were not everything to be desired? As these differ markedly with the different sinuses it will be best to consider them from their separate and distinct standpoints.

FRONTAL SINUS.

It is presupposed in this sinus that an external operation through the brow with removal of one or more of the walls has been performed, what untoward sequelæ of a more or less permanent nature may follow?

1. Persistence of the pain.
2. Hemianesthesia of the brow and scalp.
3. Persistence of the discharge.
4. Neuralgia about the cicatrix.
5. Diplopia.
6. Epiphora.

I am purposely omitting the severer forms such as meningitis, blindness, fistula formation, etc., as our discourse is limited to the minor conditions which, however, are of sufficient annoyance to the patient as to prevent the operation from being termed a complete success. Patients are only too prone to forget the previous severity of their disease and dwell upon some present condition which would have been considered trivial had they suddenly been transformed from one to the other, but after all it is only human nature and there is not one of us here that would not feel similarly disposed were the personalities reversed and we were the patient instead of the physician. When a patient undergoes an operation he expects to be cured and all cured and should some slight trace of the old trouble linger or a new symptom appear, it is bound to be a source of dissatisfaction, and very real dissatisfaction at that, but if we could only make them realize that the surgical intervention was the means of saving or prolonging their lives or at least rescuing them from a state of chronic invalidism and they could also be made to understand that the chances of complete recovery are always inversely and not directly to the extent and severity of the

operation, the surgeon's task would be far easier than as at present constituted. To revert then to the untoward sequelæ let us for a moment consider the first one, that of persistence of the pain. After an external operation on the frontal sinus, if the pain persists, to be perfectly frank, either the operation was incompletely carried out and a focus of infection permitted to remain or the pain is due to some other sinus or, indeed, to some other condition which may be quite remote. I recall distinctly two patients which my operation failed to cure of their headaches, although both had chronic sinusitis; one subsequently was permanently relieved after his acidosis had been eliminated, while with the other the gynecologist succeeded wherein I had failed. So in these cases where pain is the principal complaint, while the external operation will, in the vast majority of instances, bring about complete relief, nevertheless the prognosis should not be entirely Utopian in its promises.

Hemianesthesia of the Brow and Scalp.—This condition always manifests itself immediately following the operation due to the severance of the supraorbital nerve close to its exit from the foramen and is always mentioned by the patient more, it is true, in the order of passing rather than an actual complaint. Fortunately the anesthesia is of temporary duration as in the course of a few weeks' regeneration of the nerves or a collateral anastomosis occurs with a return of full sensibility to the previously affected area. I have never seen a case where this has become permanent.

Persistence of the Discharge.—If I were asked to name the most frequent untoward sequelæ of an external radical on the frontal sinus I would unhesitatingly chose this one. In the natural course of events a purulent discharge always follows, which gradually assumes more and more a mucoid character, at the same time becoming less and less until in the course of weeks it ceases altogether. Unfortunately in a great many cases this discharge continues much longer than we care to see it despite appropriate treatments. If, in the quiet of our study we review our operation, small wonder will it appear that such a secretion makes its appearance, for it represents nature's attempt to throw off those portions of tissues which have been devitalized by our curettes and cutting for-

ceps. When we consider the bony cell walls, periosteum and adjacent mucosa which has been crunched, scraped and lacerated together with the ravages of the disease, much regeneration of tissue must occur before these parts again assume their earlier functioning activities. The persistence of this discharge will be directly to the extent of the surgical application and should this have embraced large and remote areas, just that much longer will it require before complete regeneration occurs, if indeed, it ever does. In any event, to use a classic phrase, a *locus minor resistencia* is established which may manifest itself with every exposure to cold and wet, or indeed, any cause which would be conducive to an acute coryza. However, in the advent of such an eventuality, all criticism must be instantly swept aside when comparison is made with the previous condition of the patient. Who of us would not gladly exchange the dangers and distress of a chronic suppurating frontal sinus with inadequate drainage for an intermittent mucoid discharge with no subjective symptoms, which moreover is gradually becoming less and less with the lapse of time? I am confident we would be unanimous on this point.

Neuralgia About the Cicatrix.—In my experience this is a relatively rare occurrence and when manifesting itself is due to the severed nerve ends becoming involved in the newly formed cicatricial tissue. Under the influence of massage it usually disappears in the course of time and I have never seen a case that required a subsequent resection for liberation of the enmeshed nerve filaments.

Diplopia.—Despite the assurance of Hajek, double vision is very apt to occur when the tendon of the superior oblique is elevated and its osseous support removed unless special measures are taken to prevent it. In many of my earlier cases permanent diplopia developed especially on looking downward, as, for example, descending a stairway, which required powerful lenses to correct the defective vision. For the past several years after the completion of the operation I have caught up the loosened tendon at its point of contact with the pulley and throwing a fine catgut ligature around it, tied it to the bridge of bone approximately in its original position. Since practicing this procedure, diplopia has manifested itself in

but one case, and curiously the muscle on the operated side appeared the stronger when examined by the ophthalmologist. This was due to my placing too much tension on the tendon while bringing it into position then tying it too tight. We have now profited by this experience, and while making it snug, are careful not to put it under any tension. At this point I would like to report a unique experience which happened in the early summer, a few months ago, with the first case on which I performed a Killian operation in 1908. This girl developed a marked diplopia which obliged her to constantly wear glasses. Fourteen years later the sinus again became infected; the condition refusing to respond to conservative methods, it necessitated a reopening of the affected area. After curetting away a small portion of diseased bone the tendon of the muscle was caught and tied as in the present method. I was particularly interested to learn what effect this would have on the long standing diplopia. You can imagine my feeling of satisfaction when on removing the bandages she said, "Why I no longer see double," and subsequent examination showed that she had fused binocular vision which has remained so ever since.

Epiphora.—When one considers that the lacrimal sac is elevated from its bed and the underlying bone resected, it is to be marveled that tearing does not invariably follow the radical frontal operation. As a matter of fact, it usually does if one takes the trouble to make a test, but fortunately it seems to be of but temporary duration and disappears shortly after the patient leaves the hospital. I have referred patients to the ophthalmologist immediately following the operation, who reported that it was difficult to force a passage for fluids through the membranous canal. On reexamination in three or four weeks, syringing succeeded with no difficulty whatever showing that nature had restored to the physiologic function of the canal as a drainage passage for the tears into the nose.

Now to return to our subject of end results, how many patients are troubled with these unpleasant sequelæ and how great is their degree of discomfort? I am not prepared to give definite statistics, but certainly less than 10 per cent have any of these one year after the operation. The most frequent

is the tendency toward catching cold with a discharge, but even this in time becomes less noticeable and for all I know ceases entirely. We do not perform the radical operation (except in a few instances such as threatened cerebral or orbital complications) until all conservative means have failed; but when indicated, it is one of the most satisfactory methods of relieving and ultimately curing a severe case of chronic frontal sinusitis that we have at our command.

MAXILLARY SINUS.

The radical operation on this sinus when properly performed is seldom followed by unpleasant after effects. Certain false steps, however, may be the cause of one of the following:

1. Anesthesia of the upper lip and teeth on the operated side.
2. Permanent fistula formation into the mouth.
3. Excessive dryness of the nose on the affected side.
4. Gradual return of the discharge after an apparent cure.

Anesthesia of the lip and upper teeth. The loss of sensibility of the lip due to cutting the inferior branch of the infra-orbital nerve frequently occurs but is only of temporary duration as full sensibility soon returns. Anesthesia of the teeth is a more serious matter and should never occur as it is due to faulty technic. Permanent anesthesia of the teeth means that the nerve has been injured or destroyed and usually presages the ultimate loss of the teeth except under very favorable circumstances in the hands of a first class dentist.

Permanent fistula formation into the mouth. This unfortunate sequela usually results from making the incision too far down on the gums, thus not having sufficient redundant tissue on closure of the wound. There is no necessity for this, and it can always be avoided provided, of course, that a previous fistula did not exist. As the permanent closure of these fistulas is almost a chapter in itself, time is insufficient to enter into that phase of the discussion.

Excessive dryness of the nose and affected side. A rare eventuality unless one is prodigal in removing the soft tissue of the inferior turbinate. I have rarely found it necessary to remove much of this structure and now our routine pro-

cedure is to remove only that portion of the lower edge anteriorly that obstructs free aeration of the sinus after the counter opening is made into the nose.

Gradual return of the discharge after an apparent cure. This disappointing result is usually due to insufficient removal of the diseased lining membrane of the sinus in which permanent pathologic changes had occurred. In my experience this omission takes place in the prelacrima recess, which can only be reached by a right angle curette. It is surprising while using this instrument to note the large amount of polypoid tissue that can be contained in such a comparatively small area. Another cause of failure is making too small a counter opening into the nose. I believe for the best results this opening should extend from the anterior end of the turbinate to about its middle and from its insertion well into the floor of the nose. It must be remembered that not only drainage, but aeration is required and I question very much whether the latter is not as important as the former in healing these old antral suppurations.

SPHENOID SINUS.

The important sequelæ which are connected with this cavity are:

1. Gradual closure of the opening before the suppuration has ceased.

2. Reinfection with intermittent suppuration.

Gradual narrowing with incomplete closure of the opening is observed in practically every case following operation on the sphenoid and is undoubtedly an effort of nature to restore the parts to their original condition. In most cases this is an advantage rather than a disadvantage if we presuppose that the sinus possesses some physiologic function.

Reinfection with intermittent suppuration is not an infrequent occurrence and usually succumbs readily after a slight enlargement of the ostium, which at this time is but a minor office procedure.

ETHMOID LABYRINTH.

The postoperative behavior of these cells depends upon so many factors that this phase of sinuology represents

today perhaps the most important in the entire category of paranasal affections. New methods of operating have sprung up here and there and been hailed as the last word, only to die away and fall into discard or become so modified as to entirely lose their original identity. This fact alone shows that of all the methods hitherto devised none have proven a vade mecum for all classes of cases. How many of us have seen cases of rather mild and decidedly bearable ethmoiditis made almost unbearable and well nigh hopeless by injudicious or incomplete operations. I know of no sinus condition where it is easier to advise operation or more difficult to extricate ourselves after this badly advised piece of surgery has been consummated than this very condition. How often has an anatomically perfect, though mildly infected labyrinth been transformed into a disorganized suppurating mass, which defies the best efforts of even a master to change it back to its former morphologic configuration with its normal physiologic activities. A great many more times even in our own practices, I dare say, than we would care to admit. Let us then consider more in detail the end results often following partial and complete ethmoidectomy with certain reasons pertaining thereto.

1. Continuance of the discharge.
2. Continuance of the pain.
3. Partial occlusion of the nostril.
4. Ocular symptoms which did not obtain previous to the operation.

Continuance of the Discharge.—Of all the most operative conditions one is called upon to treat this one I believe comprises more than all the rest combined. Accepting this statement at its face value, what reason can be attributed for the prevalence of the discharge following operation? In answering this question we can, as the Chinaman says, talk "goody goody" or tell the truth. If we choose the more difficult way we will say it was because we did not eradicate the disease at the time of operation and furthermore broke into healthy tissue which also became infected so that the end result was worse than the original condition. In analyzing such a case it was not altogether our fault for we proceeded according

to our best judgment, removing the cells as they appeared to be diseased. It is just here, however, that we made our error, both in judgment and in technic for one can differentiate by inspection through a nostril, diseased ethmoid tissue which might regenerate and that which imperatively must be removed. I think the preliminary resection of the middle turbinate followed by appropriate treatment and careful observation will very largely solve our ethmoidal problem.

Continuation of the Pain.—By this I mean headaches associated with an ethmoid infection continue both in their frequency and severity. It must be remembered that many other factors may enter here, and the hypothesis that our operation was incomplete and failed to remove the cause of the headaches may be without foundation as these pains might easily have some other genesis. In view of a more or less wide experience with this class of cases I have ceased to take these complaints as seriously as formerly, and now look for extrinsic causes (if I am convinced the ethmoid is not at fault) usually with success.

Partial occlusion of the nostril is only mentioned in passing and is due to carelessness in permitting synecia to form, thus interfering in a greater or lesser degree with the respiratory air current passing through the nostril.

Ocular symptoms which did not prevail previous to the operation. This is fortunately a very rare occurrence, even though only the thickness of a sheet of paper separates the orbital fat from the ethmoidal mucosa. I have frequently seen the conjunctiva become so engorged that it protruded between the lids following extensive operative measures on the ethmoid only to again recede and finally disappear without leaving any untoward effects.

Patients occasionally complain of a weakness of the eye, but this must be classed in the nature of a neurosis for the ophthalmologist always returned negative reports. I do not recall ever having seen a case of serious disturbance of vision following an intranasal operation, although such cases have occasionally been reported. In reviewing our ethmoidal work I am sure most of us will agree that it is best to make haste

slowly and not try to cure the patient in one full swoop. It is far wiser to remove the middle turbinate as a preliminary operation not only for the purpose of permitting better aeration and the furtherance of drainage, but what is even more important to allow comprehensive study of the labyrinth, which was an impossibility with this structure in situ. Finally let us remember that experience has taught us radical operations upon the accessory sinuses do not always spell radical cures.

XI.

TREATMENT OF OTITIS MEDIA WITH TUBERCULIN.

BY G. THOMSEN VON COLDITZ, M. D.,

CHICAGO.

We read the opinions of different men every day stating how long a suppurating ear should be discharging before we may pronounce it chronic and how long a chronic ear may discharge before operative procedure is the only hope of a cure for the patient. I do not wish to argue the question of when a case is acute or when it is chronic, that is of too little importance. But as far as putting a time limit on an old chronic case—whether it should be operated at once or given six weeks' treatment and then operated, if not cured—is wrong. If an ear which has been discharging from five to fifteen years will remain dry for one to two years following a course of treatments over a period of one to two years, we can claim to have cured the ear. The patient has been benefited far greater by not operating, saving him time and money, pain and discomfort, not to mention the danger connected with the operation and the prognosis of hearing.

It cannot be emphasized too strongly that we should not only make a diagnosis of what ails a patient but find the cause. The diseased organ frequently has bacteria present, but is also affected by toxins from bacteria in some other part of the body presumably healthy. The chief mistake made in these cases is the same as it always has been and always will be as long as cutting instruments are made, that the diseased tissue, whether the primary offending organ or not, is removed and the patient pronounced cured, without having removed the cause.

We read in text books and in medical journals that the removal of tonsils will cure most anything from a tubercular keratitis to a suppurative otitis media. The removal of tonsils and teeth is as popular today as the removal of ovaries was twenty years ago. The other day a doctor showed me a pair

of tonsils he had taken from a seven year old child. They were small and pinkish—as normal looking as any I have ever seen. On asking the indication for operating, he said that the child breathed through its mouth, had sore throat once in a while, poor appetite and that the mother had had peritonsillar abscess twice. The latter alone, I am sure, justified the operation.

The medical profession today is altogether too pessimistic regarding the use of tuberculin, which certainly is not warranted by the excellent results obtained. On asking different doctors about their experience with tuberculin, I was told by more than one that it was no good. However, at the same time they confessed they had never used it. Two reasons probably responsible for this skepticism in regard to tuberculin are: first, that men do not know which test is indicated in a given case, and second, that they do not understand how to use it therapeutically.

It is true that a large percentage of patients will react to tuberculin, even though they have not an active process. This is due to the extreme delicacy of the test, and also to the fact that most people have, at some time or other, had a tubercular infection. But the answer is—you must know how to interpret the reaction and how to differentiate the active from the latent infection.

When a patient comes to me complaining of a discharging ear, I first get a careful history. Very often I find that one or both parents have died from tuberculosis when the patient was a child and that their discharging ear dates back to that time. Following the regular physical examination of the ear, nose and throat, the pus is examined microscopically and a tuberculin test is made. X-rays are taken when practical. If the patient reacts positively to the tuberculin test, the chest is also examined.

First of all let us consider the various tuberculin tests, for, as previously mentioned, it is most important to know which one should be used in a given case. I will mention them in order as to their diagnostic value:

1. The Moro Test.—This, as you all know, is a salve made of equal parts of old tuberculin and anhydrous lanolin. A

small amount of the salve, about the size of a pea, is rubbed into the skin, which previously has been washed with soap and water. I usually choose the upper part of the abdomen. When positive, within twelve to twenty-four hours there is a reaction at the site of application—a redness of the skin with papule formation. The intensity of the reaction varies in different cases, often not starting until the third or fourth day and lasting, usually, from five to ten days. Frequently, especially in so-called “scrofulous” children, the reaction is very severe accompanied with considerable itching. The advantage of this test lies in its easy application, however, it should not be used in children over ten.

2. The von Pirquet Test.—This, like the Moro test, is of value only in children. The technic consists in scarifying the skin, choosing the inside of the upper arm, which has been made sterile, in two places about an inch to two inches apart. Care should be taken so as not to draw blood. A drop of old tuberculin is now placed on one of the scarified areas, the other being used for control. If positive, an indurated area appears around the spot where the tuberculin has been applied. It is the tuberculin coming in contact with the epithelium of the skin that causes the reaction. The reaction varies in degree according to the susceptibility of the patient and the amount of tubercular infection present. We may have a local reaction at the same time in an eye, for instance, with a tubercular iritis present.

3. The Conjunctival Test.—This consists of the instillation of a drop of one per cent Koch’s old tuberculin solution into the conjunctival sac. The eye is allowed to close for a minute and then the lower lid held down for a minute. Within eight to twenty-four hours we get a reaction which may only consist in a redness of the lower lid, but may also be most severe, causing inflammation of the uveal tract, conjunctiva bulbi, and edema of the lids. The reaction may be so severe as to injure the eye, and, therefore, should not be used.

4. The Intracutaneous Test.—This test is very reliable and may be used in children as well as in adults. Besides being of a positive diagnostic value, it has the advantage over the subcutaneous test, which is considered by many as the most valuable one, in that it may be used in ambulatory cases. It

has the advantage over the other tests mentioned previously in that we know the exact amount of tuberculin used, which amount may be increased in consecutive tests.

The technic is as follows: A fine needle is best adapted for this purpose, with a regular tuberculin syringe. A fresh solution of old tuberculin, the strength of which is one milligram to one-tenth of a cubic centimeter of sterile water, is drawn up into the syringe, which has been sterilized. The forearm is now made sterile and the needle introduced into the stretched skin, running the same parallel for about a quarter of an inch. Then we inject one-tenth of a cubic centimeter into the skin in such a manner that a white bleb is formed, showing it to be intracutaneous. The needle is then carefully withdrawn. When positive, a slight reaction is seen within six hours and a marked reaction within twenty-four hours, a zone having formed around the injection, the size of which indicates the amount of reaction. The halo may reach the size of a silver dollar, but the average size is about that of a nickel or a quarter. The reaction is most pronounced at the end of the second day, and on the third it begins to diminish. Reaction may be present for several weeks, the periphery being the first to disappear. If no reaction has occurred in three days, we inject in similar manner three milligrams, and three days later, if negative, five milligrams. Some men go as high as ten milligrams. Personally, I have never given over five milligrams in a suspected case.

5. The Subcutaneous Test.—This test is without a doubt the most valuable one we possess, although its reaction is the most severe and is, therefore, contraindicated in certain cases, in which instance we should use the intracutaneous test. One great advantage of this test is that in a positive case we not only get a local reaction but also a focal reaction which is most valuable. The procedure for this test is as follows: The patient should be kept as quiet as possible, not necessarily in bed, for two days prior to the injection of tuberculin, in order to get his normal temperature and the range of same. The temperature should be taken every two hours and a careful record kept. On the third day the tuberculin is injected, preferably in the morning so as to be able to see the reaction within six hours. The patient is kept at home but not neces-

sarily in bed. The writer favors Koch's old tuberculin. The injection may be given into the arm, leg or abdomen or almost any other practical place. Usually the upper arm is found to be the most satisfactory. The regular Parke-Davis tubercular glass syringe with a platinum needle is the most practical and is easily kept sterile. According to Koch, three injections at intervals of three days are given, the first injection of 1 mg., the second of 5 mg., and the third of 10 mg. Personally I consider this dosage a little high and give 1 mg. for the first, 3 mg. for the second and 5 mg. for the third dose at three day intervals. Naturally these injections are given with all aseptic precautions. The patient is instructed to keep quiet and not scratch the arm or put on cold compresses to relieve the reaction. The temperature is taken regularly again and compared with the temperature before the injecting. In positive cases we usually get a reaction within ten hours, but it may start within six. The temperature will vary from 100 to 102 degrees F. and over when the test is positive. The first injection may give a slight reaction and the second a very severe one; in that case a third injection is not indicated.

Inasmuch as the temperature plays a most important part in this test, it would be appropriate to say a few words about taking the temperature. Most thermometers are marked either one-half or one minute and are usually only given that amount of time for taking the temperature. The average doctor does not even seem to take into consideration whether the patient has just come in from the cold or just had a glass of ice water to drink, not to mention the nurse who seems to think that speed is essential in taking temperature. Rarely do I find a patient whose temperature will go to normal in one minute with a half minute thermometer. To be sure of an accurate temperature, a thermometer should be left in the patient's mouth for five minutes and then read and put back in the patient's mouth for one minute. If the temperature remains the same, it is correct; if not, repeat until it does not rise. I have left the thermometer in the patient's mouth for ten minutes before getting the maximum temperature. The old fashioned method of taking the temperature by axilla, I believe has been entirely discarded. The rectal

temperature when possible should always be used by preference as this is the most dependable and will, at times, show elevations of temperature which will not be shown by mouth. This is particularly true in mouth breathers.

The writer wishes to state here that he has had a number of cases of old chronic suppurating otitis media, some of five to fifteen years' standing, that have gone from one doctor to another, have had many treatments and were then told that unless they were operated upon they would either not be cured or would die from meningitis. These same cases, after careful examination gave a ++ and +++ tuberculin reaction, received local treatments combined with graduated tuberculin injections and were absolutely cured within six months to a year and a half. In some cases the hearing was restored to normal after the perforation in the tympanum had been closed with subsequent treatments. At the same time the patient's general health was markedly improved.

Of all adults treated only one showed tubercle bacilli in the pus, together with other bacteria. This patient, when I first saw him, besides having a suppurative otitis media, was in the last stage of pulmonary tuberculosis with involvement of the larynx. He claimed his ears had been discharging for years. The patient died within about a month after the first time I saw him from general tuberculosis. His treatments were only general. As expected, in this case there was no reaction following the tuberculin test.

The writer has had a number of cases other than ear that he would like to just touch upon in connection with this tubercular treatment to show how commonly tuberculosis is overlooked and often diagnosed as syphilis.

One patient, with a large perforated septum was pronounced by an examining specialist as syphilitic. I made a tuberculin test and it reacted ++. After tuberculin treatments the nose cleared up from crusts and discharge, and the patient was comfortable. She considered herself cured.

Another patient was treated for syphilitic vocal cords which were almost gone. He could not speak above a whisper. The tuberculin test, intracutaneous, showed +++ reaction. After 20 B. E. tuberculin injections the cords appeared normal

and the patient had a strong voice again so he could do public speaking.

A third patient was treated for syphilitic keratitis by an eye specialist. After 20 B. E. tuberculin injections the eye was well. The cornea which had been so opaque that the iris could not be seen was clear and the vision good.

I am aware of the fact that many doctors claim the same results that are obtained with tuberculin can be obtained with yeast and other proteins, but personally I am convinced that this is not correct, basing my statement on the results I have obtained in various cases. The statement that all patients over a certain age respond to a tuberculin test is also contrary to my findings.

I do not mean to claim to be able to cure all suppurative otitis media cases, but I wish to call the attention to the fact that many cases are neglected that could be cured with tuberculin.

The following cases came to me for treatment of chronic suppurative otitis media. I am only considering those which reacted positive to a tuberculin test and that were treated with tuberculin in addition to local treatment, in which cases I attribute the good results to the tuberculin used.

REPORT OF CASES.

1. Ruth U., age 20, single, American, Swedish parents: Family history negative.

Personal History.—Patient had diseases of childhood. For past three years patient has been getting boils on arms and her back, new ones starting every few weeks to a month. Some boils contained as much as two drams of pus. Had several courses of autogenous vaccin, with no results. Was then put on diet with yeast, no results. Then one year ago patient's right ear started to discharge.

Examination.—Patient well developed, well nourished. Chest $30\frac{1}{2}$ inches, $1\frac{3}{4}$ inch expansion and negative. Temperature 99.4, pulse 82. Number of boils on arms and one very large one on back. Right ear: Mastoids normal. Tympanum, small central perforation with slight discharge. Pus shows staphylococci. Tuberculin test was made; 1/10 cc. representing one milligram of old tuberculin was injected intra-

cutaneously. In twenty-four hours there was a very marked reaction, the halo reaching the size of a silver dollar and lasting for over two weeks. This was what I term a +++ reaction.

Treatment.—Patient was given B. E. tuberculin injections, one every five days, receiving thirty injections in all. After about fifteen injections, the boils disappeared and the ear was dry. There has been no recurrence. Patient gained about ten pounds in weight. There was considerable reaction following each injection.

2. Miss A. R., age 36, American, German parents.

Family History.—Father died at 38 of tuberculosis, sick three years. Mother died at 44 of tuberculosis, sick six months. One sister living and well, and one brother has ear trouble.

Personal History.—Patient had whooping cough, diphtheria, chickenpox, measles and mumps as child. When 10 years old, right ear started to discharge, getting more profuse each year. At the age of 16 started headaches over frontal sinuses, which she has had the greater part of the time. For the past ten years patient has had a slight cough, raises a great deal of mucus mixed with black particles. Three years ago pain developed in the left knee. She was unable to straighten out her leg at night on account of pain. X-ray pictures of the knee were negative. In spring of 1920 left ear started to discharge. Patient perspires slightly at night.

Examination.—General condition good. Height 5 feet 2 inches, weight 116 pounds. Temperature 99, pulse 80. Chest negative. No enlarged glands. Mastoids: External inspection negative. Auditory canals large. Right tympanum only small margin left. Left tympanus medium sized perforation. No reflex. Retraction of malleolus. Both eustachian tubes free. Right ear discharge very profuse, left ear small amount. Hearing almost normal. Pus microscopic: Diphtheria and diphtheroid bacilli. X-ray showed no cells in mastoids and cloudy frontal sinuses. Tuberculin intracutaneous test gave a +++ reaction.

Treatment.—Started April, 1920. Auditory canal closed with cotton on applicator. Ears inflated with iodine, menthol and camphor and autogenous vaccin given over a period of

two months, no results. In October of the same year treatment continued. This time B. E. tuberculin used. Treatments again extended over two months. Discharge decreased, patient's cough diminished and knee felt better. The following June treatments were continued for one month. Left ear stopped discharging. Knee was entirely free from pain: no more cough, and headache gone. Slight discharge in right ear. January, 1922, patient returned for treatments with only very slight discharge in right ear. Left ear dry. No headache, no cough, no expectoration. Knee normal. There was reaction after every injection.

3. Miss E. A., aged 20 years, American born.

Family History.—Parents both dead. Patient believes mother died of tuberculosis. No brothers or sisters.

Personal History.—Patient had diseases of childhood. Coughs a little, especially in morning. Has night sweats once in a while. Discharge from both ears since childhood. Has had treatments at different times. Feels tired all the time. Great deal of headache.

Examination.—Tall and poorly nourished. Anemic. Enlarged superficial cervical glands. Tonsils No. 11. Temperature 99.3 degrees F. Eyes: Slight conjunctivitis. Mastoid prominent, not tender: large perforations. Rinne test negative. Eustachian tube free. Hearing: Watch R. E. $\frac{1}{2}$ ft., L. E. 1 ft.

Treatment.—Ears kept clean and packed with dry gauze. T. R. tuberculin injections every five days. At end of ten months' treatment, both ears dry. Patient left the city after that and went to Michigan. About one year later I heard from a friend of hers that she was married and in good health.

This patient had a Moro test made and reacted strongly to the same. This was in 1915, when I was not using the intracutaneous test, to which I now give preference.

4. J. M. H., age 33 years, male, married; born in Norway. Family history negative.

Personal History.—Patient always well, except discharge from right ear for the past seventeen years. Has had treatments without results.

Examination.—September 8, 1915. Patient well developed. Chest negative. Left ear: Normal in appearance. Hearing normal. Right ear: Mastoid normal. Auditory canal negative. Tympanum, large central perforation. Thin purulent discharge containing staphylococci and streptococci. Hearing: Watch at $\frac{1}{2}$ foot. Moro test negative.

Treatments.—Ten injections of stock vaccin given in connection with local treatments with no improvement.

After a period of about one month, T. R. tuberculin injections were given, one injection every five days and local treatments every three days. A slight reaction followed each injection. The ear would stop discharging for a few months, then when the treatments had been discontinued for a while the ear would start discharging again. At the end of eight months the ear remained dry. After that local treatments were given to close the perforation in the tympanum, which was accomplished in three months. Hearing two years later was watch at 4 feet. The ear had remained dry.

5. M. P., aged 15 years, female, American born, Italian parents.

Family History.—Father killed by blackhand, mother well. Five sisters and five brothers living and well. One sister died from infected axillary gland.

Personal History.—When patient was two years old the right ear started to discharge. One year later mastoid operation performed by the writer. Some time later the left ear started to discharge. The patient complains of a great deal of headache. Always complains of not feeling well and being tired. The mother has remarried and the patient gets no attention at home.

Examination.—Chest very large, overdeveloped breasts, otherwise negative. Enlarged superficial cervical glands. Ears: No tenderness over mastoids. Very large tympanic perforations. Profuse discharge from both ears showing staphylococci and pneumococci. Hearing: R. E. watch 1 foot. L. E. watch $\frac{1}{2}$ foot. Rinné test R. E. —, L. E. —. Temperature 99.3 degrees F. Moro test negative; 1/10 tuberculin intracutaneous gave little reaction; 3/10 tuberculin injection gave marked reaction.

Treatment.—Ears cleaned with cotton. Patient told to keep cotton in ear, but comes to office without it every time and says, "I forgot." No other treatment given except tuberculin injections. The patient has now been treated for $1\frac{1}{2}$ years. After about one year's treatment, which had been interrupted during the summer, the right ear has remained dry for six months and the left ear has only a very slight discharge.

This patient's results have been particularly gratifying, inasmuch as she has been so irregular in coming for treatment, and has taken no care of herself at home to assist in a cure. Her physical condition in general has also improved during these treatments.

CONCLUSIONS.

1. A patient with a chronic suppurating ear should always be tested for tuberculosis.
2. When a patient reacts positive to the proper tuberculin test, he should be treated with tuberculin, even though other bacteria are found present in the ear discharge.
3. A patient reacting positive to a tuberculin test should be impressed with the fact that his prognosis is good, providing he takes his treatments regularly.
4. A mastoid operation should be done only as a last resort for cure.

XII.

THE PRACTICAL DIAGNOSTIC VALUE OF TESTS OF THE VESTIBULAR MECHANISM.

BY FRANK L. DENNIS, M. D.,

COLORADO SPRINGS.

In view of the increasing use of tests of the vestibular apparatus and the numerous discussions of the subject appearing in the literature, is it not time to take stock of our knowledge and review some of the reports made?

Examination of the vestibular mechanism is already of tremendous usefulness in diagnosis and is destined to be of still further service as we add knowledge to our store from the labors of keen workers in this field. I need only mention among others, the recent contributions of Ingham and Jones, Fisher, Bárány, Neumann and Buys. A host of questions arise in the minds of those doing this work:

What effect does epilepsy have on the vestibular mechanism?

Is there a toxemia in tuberculosis that causes definite reactions?

In what manner does encephalitis affect the responses, and why?

Is there an orientation center in the frontal lobe, as suggested by Lasagna, and what relation has it to the pathways from the labyrinth?

Why does one occasionally see nystagmus in the early stages of pneumonia and erysipelas?

When these questions and others are answered, the diagnostic value of these examinations is bound to be enhanced.

As is inevitable when any new procedure is instituted, variations in technic creep in, some of the claims made in the beginning are not substantiated, others are strengthened, and, piece by piece, the structure of fact is built up. It is the purpose of this paper to call attention to some of these details and to record some observations which, it is hoped, will contribute to the sum total.

In the first place, I desire to emphasize the need for a uniform technic in conducting the examinations and a uniform nomenclature in recording the findings. One observer douches with water at 68 F., another at 65 or 55 F.; in one case, rotation is carried on at the standard rate of ten turns in twenty seconds for the production of nystagmus, while in another the rate may be greater or less, and so on. Such variations may or may not be important, but surely, in establishing the value of any procedure where so many points are still undetermined, uniformity of technic is essential.

In some reports, many facts are not stated; such as, for instance, the exact state of the hearing; the number of past pointings after rotation; measurement of vertigo after stimulation; whether there was nausea and vomiting following the test; or whether the complete test was made at one sitting, and, if not, the number of seances required to complete it.

Care is important. A faulty position of the head in turning or douching may cause apparent perversion or inversion of the nystagmus, as pointed out by Jones and Fisher.

Let us follow closely the standard technic of ten turns in twenty seconds for the production of nystagmus, ten turns in ten seconds for the measurement of vertigo and the eliciting of past pointing, and douching with water of exactly 68 F. for the caloric test.

In this connection I refer to a most excellent paper by Vail¹ on the results of the vestibular tests in ten cases of acusticus tumors. It is worthy of note that the last three cases of this series exhibit the typical "angle lesion syndrome," because they were so examined as to bring this out, while the earlier cases were not so examined and do not show it. The one additional thing which would have completed the picture is a caloric test of the horizontal canals of both sides. Undoubtedly, all the available data were reported by Dr. Vail, but the point is that, in all probability, when the earlier cases were examined, the significance of all the factors in the angle lesion syndrome was not appreciated. In but few of the cases was a measurement made of the vertigo response, and in the caloric tests the temperature of the water varied between 50 and 68 F. According to Jones and Fisher, angle lesions produce the most typical vestibular responses of all intracranial

lesions. The latter, in a personal communication, says that we can be more definitely sure of the diagnosis when this syndrome is present than with any other combination of findings.

Intracranial lesions are notoriously difficult of localization on account of variable responses due to pressure. Pressure affects remote parts of the brain as well as near by areas, and it requires nice judgment to say when a given finding is due to pressure, distant or near by, and when it is due to the lesion itself.

We wish to learn from these tests (1) whether the lesion is central or peripheral; (2) whether the lesion is toxic (evanescent or permanent), and (3) if central, where it is.

Many times, the results of neurootologic tests will answer these questions. Sometimes, indeed, they only will give definite answers. In every case, the results of the tests will be helpful in arriving at a diagnosis, if studied, as they should be, in connection with neurologic, ophthalmologic and serologic findings. In the present state of our knowledge, we must be content to confine our localizations of intracranial lesions to supratentorial or infratentorial, and right or left side, as the case may be. This alone is no mean help in a great many cases. Occasionally, the findings may permit us to infer a more accurate diagnosis. In not a few instances the vestibular findings have been the deciding factor in the question as to whether to explore the posterior or middle fossa, and sometimes, when their significance has been opposed to the indications from other examinations, subsequent events have justified their accuracy. Greater knowledge and experience will, in all probability, permit a more minute localization.

It is important that the examinations be made by one trained in otology. On the other hand, it is most desirable that the interpretations be worked out with a neurologist who is familiar with the findings of vestibular tests.

We have come to believe from past observation that certain things have a definite significance: Spontaneous vertical nystagmus upward is indicative of a central lesion; after stimulation, conjugate deviation of the eyes (lack of the quick component of nystagmus) and exaggerated responses point to a supratentorial lesion, as exemplified by Lyons' report of nine cases of supratentorial tumors, diagnosed independently by

the Bárány tests.² All were proven cases, either by operation or necropsy. Perversion of nystagmus means trouble in the brain stem, either from a lesion there, or indirectly, from near by pressure. Loss of vertigo responses suggests disturbance in the cerebellum; and the so-called "angle lesion phenomenon complex," consisting of deafness and total failure of vestibular responses on the affected side, together with, on the opposite side, failure of the verticals' responses but with good responses from the horizontal canal, has been found so often in proven angle lesions as to be regarded as a most important diagnostic sign.

It has been the experience of all who do this work to encounter loss of responses from the vertical canals in cases in which there was probably no organic central lesion. A strict application of the theory of neuraxial differentiation of the pathways from the verticals and those from the horizontal canals, as advanced by Jones and Fisher, would force us to conclude that these are instances of central lesions, pontile in location. However, the matter is not so simple. It is quite possible to explain this phenomenon without invalidating that theory. It is believed that toxins can select certain parts of the nervous pathways to the exclusion of others for the exercise of their deleterious action, the vulnerability of the cochlear branch being well known. We know that, even in cases of central lesion, distant pressure can ablate the responses from the verticals and not so affect those from the horizontals. The responses from the vertical canals are much more easily affected than those from the horizontals—possibly because the latter, in our daily activities, are used so much more than the former. It is also a fact that nystagmus, after the turning of normal persons, is much shorter in duration from the verticals than from the horizontals. In a series of examinations of 550 aviators which I made at Rockwell Field, the following averages were noted:

Turning to the right ten times in twenty seconds with head 30 degrees forward, nystagmus of 22.36 seconds' duration; turning to the left, nystagmus of 23.44 seconds' duration. These results agree closely with the averages obtained in 75,000 successful candidates for aviation, as tabulated by the Air Medical Service, their figures being 23 and 23.1 sec-

onds for turning in the two directions, respectively. One hundred and eighty of these men were rotated with the head 60 degrees backward, thus stimulating the anterior vertical canals. The findings were: Turning to the right, the nystagmus time averaged 15.56 seconds; turning to the left, nystagmus time averaged 16.19 seconds.

The posterior verticals were tested in 300 men, by rotating them with the head on the right shoulder. The average nystagmus time after turning to the right was 16.85 seconds; after turning to the left, 15.60 seconds. It is, of course, understood that when we speak of stimulating the posterior or anterior verticals, this is true only in a certain sense, and that when one vertical canal is stimulated, the other also participates.

These experiments prove that the average nystagmus time after turning is decidedly less from the vertical canals than from the horizontals. This fact may have some relation to the fact that the verticals are more vulnerable. Owing to this greater vulnerability of the vertical canals and their pathways, much care should be exercised in evaluating the importance of failure of their responses.

Anomalies of past pointing after stimulation are also frequently encountered. This may be due to the fact that past pointing is a voluntary act. It is a manifestation of vertigo, and vertigo, being subjective, can be controlled in its manifestations by education and practice. My own experience leads me to place less reliance on aberrations of past pointing than on any of the other findings from vestibular tests, and to disregard them unless they are corroborated by other findings indicative of central lesion.

Discussion and data on the foregoing two points are much needed to make clearer their significance. Much good judgment is needed in weighing all the evidence adduced in these tests, and in all cases it should be studied in connection with the findings from all other examinations.

One of the difficult things to interpret is the changing pictures which we get on reexamining certain cases. I do not refer to the changes which take place in a case in which a tumor is growing in a certain direction, and thereby involving different pathways and thus adding to the signs. I refer

to those cases in which there is a return of the responses which were previously lacking. Ingham and Jones, in their paper presented recently before the American Otological Society, describe such a case. I reported a similar one at the meeting of the Midwestern Section of the American Laryngological, Rhinological and Otological Society this year, and Case 8 of the series reported in this paper exemplifies it. These experiences emphasize the need for repeated examinations and careful consideration before formulating conclusions.

In the following reports, cases have been purposely selected, with one exception (Case 10), in which the diagnosis has not been confirmed by operation or necropsy. Many of them present difficulties in diagnosis. They are divided into three groups: Epilepsies, toxemias (including tuberculosis), and intracranial lesions. The group of syphilitic toxemias is omitted. All have been examined with the routine technic outlined above, except when variations are noted.

REPORT OF CASES. FIRST GROUP, EPILEPSY.

Case 1.—Mrs. L. V. S., aged 59, examined May 10, 1921, gave a history of measles and whooping cough in early childhood, abscess in right ear at 7 years, and diphtheria at 12 years. She had been a little deaf since having the abscess, growing worse for the last eight years.

For ten years she had been having nervous attacks every three or four weeks; the eyes turned in; the right arm jerked and twisted inward, sometimes the head twisted and she became unconscious. Immediately preceding the attacks she had a sensation as if something flashed by her head from right to left. She did not bite the tongue or suffer nausea during attacks. She thought she was dizzy at times. She had no headaches. The attacks, which lasted from three to five minutes, were diagnosed as epilepsy by a neurologist.

Examination of the nose and throat was negative. Examination of the ears revealed that both drum membranes were intact and normal in appearance. The hearing was:

0	1"	poor	ac < bc	> n	6"
C ⁶⁴ — C	C ⁴ —	W. V. —			
4"	10"	good	ac > bc	= n	8'

Weber to right from left mastoid.

Vestibular examination (Fig. 1) revealed as notable points, slightly exaggerated nystagmus and correct past pointing from turning with head 30 degrees forward, but with diminished vertigo.

On douching of the right ear with the head 30 degrees forward, there was no nystagmus but correct past pointing. On putting the head back, there was good reaction from the horizontal canal in nystagmus and past pointing.

On douching the left ear, with the head 30 degrees forward, there was a very poor nystagmus, with correct past pointing elicited. On putting the head back, there was an excellent response. The entire examination was completed at one sitting. There was no nausea, pallor or sweat.

Two days later, the patient was turned with the head back 60 degrees and forward 120 degrees. In the former position a very poor nystagmus of short duration was elicited on turning to the right but none on turning to the left. In the latter position the falling reaction was correct. Douching of the left ear gave no nystagmus from the verticals, but a correct response from the horizontal canal.

Case 2.—Mrs. E. R. W., aged 39, who was examined April 20, 1921, gave a history of three family deaths (father, brother and sister) resulting from tuberculosis. The patient had had pulmonary tuberculosis ten years ago, but the case was now arrested. She had had the usual childhood diseases, and had pneumonia in early childhood. She had typhoid at 19. On getting up one night, one year ago, she was very dizzy and lost her sense of direction. She could not stand up the next day on account of vertigo, but she gradually grew better during the next four or five days. She had a second attack one month later, lasting ten days. The last attack occurred one week ago. On sitting up in bed to pull up the covers, she grew very dizzy and vomited, and she could not lie on either side. This lasted until 4 a. m., when she grew better. By noon the next day she was free from vertigo. After lunch she lay down and again became dizzy. On the following day the menses appeared and vertigo ceased. During this attack there was a slight horizontal nystagmus to the left on looking to the left when she sat up, and on lying down there was a

rotary nystagmus to the right on looking to the right. There was a history of epileptic attacks for the last seven years, occurring at intervals of from five to twelve months.

The patient had been under the care of two competent neurologists, who reported: Blood and spinal fluid Wassermann reaction negative; eye grounds negative; fields normal; reflexes normal; all cranial nerves normal; no convulsions in babyhood but one brother had them and mother and one sister are nervous wrecks.

Examination revealed: The nose, throat, teeth and ears were negative except that there were stumps of tonsils remaining. The hearing was:

3"	6"	good ac > bc < n	.7	20' +
C ⁶⁴	C	C ⁴	Galton	W. V.
2"	6"	good ac > bc < n	.7	20' +

Weber to left.

Vestibular examination (Fig. 2) revealed: No spontaneous nystagmus or past pointing; negative Romberg sign. The notable findings are: Subnormal nystagmus from horizontal canals, very much diminished vertigo; no nystagmus and only slight vertigo from right vertical canals; poor nystagmus and marked vertigo from the left verticals, correct past pointing from all canals; nausea after completion of caloric test. The examination was conducted in two sittings, with a five-day interval.

Case 3.—H. K., aged 16, second child, who was examined Sept. 8, 1921, and whose family history was negative, was a blue baby. The tonsils had been removed nine years previously. He had had chickenpox, whooping cough and influenza, and his stomach had always been easily upset. He was otherwise healthy. He had had two attacks of epilepsy, the first one nine weeks previously, the attacks following over-eating. There was no history of vertigo, deafness or tinnitus, though the patient said that the ears had felt stuffy for two days. He had a slight cold.

On examination, there were no neurologic findings; the heart was slightly irregular; blood pressure: systolic, 108; diastolic, 50. Both inguinal and right epitrochlear glands were palpable. There was considerable adenoid tissue and stumps

of tonsils remaining. The right membrana tympani was congested, the left normal. The hearing was:

	3"	12"	good	ac < bc > n
C ⁶⁴	—	C	—	C ⁴ —————
	4"	12"	good	ac > bc = n

Weber to right.

Vestibular examination (Fig. 3) revealed: No spontaneous nystagmus, past pointing to left with left hand. The entire test was made at one sitting.

On stimulation, there was subnormal nystagmus on turning to the right, markedly diminished vertigo, erratic past pointing with the left hand; no nystagmus from the right verticals and a very poor response from the left verticals; good nystagmus from both horizontals; nausea; sweat, pallor.

The findings common to all three of these cases are: Good hearing (except the first, in which the deafness is of the obstructive type), poor vertigo responses, interference with the nystagmus responses from the verticals, and correct past pointing. The findings are reported as a matter of record, and no attempt is made to draw deductions from them. So far as I am aware, no vestibular findings have previously been reported in epileptic cases.³

SECOND GROUP: TOXEMIA.

Case 4.—Mrs. McC., aged 47, whose chief complaint was vertigo, gave the following family history: The father died at 63, of cancer; the mother at 69, of pneumonia; two brothers are living and in poor health. She had earache, measles, mumps and malaria in childhood. She had been operated on for pelvic trouble fifteen years previously. She had chronic interstitial nephritis. The blood pressure was 190, systolic. Eighteen years previously she received a blow on the root of the nose, which affected the left eye, causing a blind spot. She had had dizzy spells before the pelvic operation.

She had suffered from the present illness for the last six or eight months. She had been dizzy at times, usually worse in the mornings. The left foot turned out too much, so that she struck her heels against each other. She could not walk straight. Occasionally she had tinnitus. She was subject to headaches, which are sometimes severe, and during the head-

ache had pain in the left ear. She was generally well otherwise except that her head did not "feel right." The hearing was:

C^{64}	4"	10"	good	20' +	$ac > bc = n$
— C —	4"	6"	good	20' +	$ac > bc = n$
				W. V.	

Weber to left.

Vestibular examination, in two sittings (Fig. 4) revealed: No spontaneous nystagmus nor past pointing; Romberg sign; tendency to fall to left, not influenced by position of head. On turning, there was slightly hyperactive nystagmus and past pointing with subnormal vertigo.

The caloric test revealed: No nystagmus from right verticals, nor past pointing with right hand, rather poor nystagmus from right horizontal and right hand does not past point; poor nystagmus from left verticals and good from horizontal; no past pointing; little subjective vertigo but some nausea. Patient vomited after reaching home.

One week later the ears were douched again and an entirely different picture was shown. Responses were prompt and good from verticals and horizontal of the right side, although the left hand did not past point. The nystagmus from the left verticals was delayed, and there was no past pointing. A report was made that the neurotologic tests pointed to toxemia. Under appropriate treatment of the kidney condition the vertigo disappeared.

One is puzzled to explain the change from a poor to a good reaction from the caloric test within such a short period. This case also illustrates the importance of repeated examinations.

Four patients having tuberculosis have been subjected to the neurotologic tests, all of whom complained of vertigo. All showed abnormal findings on vestibular stimulation. Two of these had middle ear tuberculosis, bilateral in one, on whom a radical mastoid operation had been done on one side. The other two had practically normal hearing. The suspicion was entertained that toxemia from a tuberculous infection may be a possibility. The record of one follows:

Case 5.—A. P. M., aged 47, came to Colorado on account of pulmonary tuberculosis, one year previously. He had little

trouble. The first attack of dizziness occurred more than a year previously, and immediately followed roentgen ray treatment of a tooth which was extracted soon afterward. A severe attack of vertigo six months before examination, with no vomiting. Another attack occurred two weeks before examination. The patient had a tendency to fall to left and backward, and objects turned to the right.

He has had pneumonia, mumps and stomach and bowel trouble. The tonsils have been removed. There was a history of a discharging ear several years previously.

Examination revealed: The nose and throat were negative. The ear drum membranes were normal; the hearing was good. Vestibular examination was made in two sittings (Fig. 5). There was no spontaneous nystagmus; the Romberg was negative; sometimes the patient past pointed to one or the other side, and sometimes touched. There were slightly subnormal nystagmus and vertigo after turning; no responses in nystagmus or vertigo from verticals of either side. Past pointing was erratic. From the horizontals, there were good nystagmus and vertigo. Douching was repeated four days later with the same results.

The significant points are lack of responses from verticals, with good responses from the horizontals and good hearing.

THIRD GROUP: INTRACRANIAL LESIONS.

Case 6.—Mrs. E. T. P., aged 48, examined May 20, 1921, complained of vertigo and nausea. She had always been well except for an operation for gallstones and diseased appendix four years previously. The first attack of vertigo occurred fourteen years previously, and next one four years previously. The last attack, occurring six weeks previously, was followed immediately by tonsillitis. The patient vomited the first day and was better the next, but had been more or less dizzy ever since. There was no headache. She had a "gripping" feeling in the back or top of the head, like pressure. She had been deaf in the right ear for eleven years. There was tinnitus at times.

Examination by her physician was negative. The spinal and blood Wassermann reactions were negative. Examination of the nose and throat was negative, except for diseased

tonsils. Examination of the ears revealed that both membrana tympani were intact; the right tube was narrow. The hearing was:

4"	8"	poor	4.0	6"
C ⁶⁴ — C	C ⁴ —	Galt.	W. V.	
4"	8"	fair	1.1	36"

Weber to left. The fork on the right mastoid was heard only in the left or better hearing ear.

The vestibular examination (Fig. 6) was conducted in two stages.

The noteworthy findings were: No spontaneous nystagmus; Romberg sign positive; falls backward with all positions of the head. After stimulation, there were subnormal nystagmus and vertigo; no past pointing with left hand after turning to left. On douching, there was no nystagmus, vertigo or past pointing, from right vertical canals, and barely a trace from verticals of left side. There was correct nystagmus from the right horizontal, and a good but perverted nystagmus from left horizontal. Ten days later the douching was repeated. At this time there was no reaction from the verticals and a questionable one from the horizontal of the right ear. Douching of the left ear showed no change from previous examination.

Eleven days afterward the ears were douched with the head 60 degrees backward. In the right ear, after five minutes' douching, there were no responses from verticals or horizontals. In the left ear an oblique nystagmus upward and to the right, of small amplitude, appeared after one minute and forty-five seconds, which became rotary on putting head upright. The patient past pointed only with the right hand to left before putting head upright.

She was referred to an oculist, who reported that there was no intraocular pathologic condition, but that the patient had 4 degrees of exophoria, and the glasses she was wearing were not properly corrected. Correct glasses relieved her of vertigo attacks.

Four months later another vestibular examination (Fig. 7) was made. The differences between the findings in this examination and those of previous ones are: A slight increase in

the nystagmus and vertigo time after turning; no past pointing with either hand after turning to the left, while at previous examinations the right hand past pointed to the left.

On douching, there was now a poor and delayed nystagmus from verticals of both sides, whereas previously these had failed to respond, and there was a good nystagmus from the right horizontal. The nystagmus from the left horizontal was still perverted. The entire examination was made at one sitting, without any subjective disturbance, except slight vertigo. The patient said that she was free from vertiginous attacks and felt well.

A report was made after the two earlier examinations that the ear tests pointed to a cerebellar lesion on the right side; and, in spite of the subjective improvement, I am still inclined to think there is intracranial trouble. The case illustrates the difficulties one meets in interpretation owing to the changing picture. The question of toxemia (possibly from a focus in the tonsils) must be considered, but it is difficult to reconcile the perverted nystagmus with this hypothesis.

Case 7.—Diagnosis by the neurologist: Encephalitis. Miss M. G., aged 18 years, on Oct. 18, 1921, complained of numbness on the left side of the face. This proved on examination to be limited to the chin area. Three days later, the patient became very dizzy, the attack lasting three days. One week later another dizzy attack appeared, accompanied by nausea and vomiting, staggering and falling to the left and tinnitus.

Examination revealed that the spinal fluid and blood Wassermann reactions were negative. Nose, throat and ears were normal; the hearing was normal. There were no pupillary abnormalities; eye grounds were negative. Adiadokokinesis of the left hand and marked incoordination of left hand were noted.

Vestibular examination two days after attack of vertigo began (Fig. 8) revealed: Spontaneous horizontal nystagmus to right and left, and vertical and rotary nystagmus on looking upward; past pointing 4 inches (10 cm.) with the left hand; Romberg sign positive.

On douching, there was no nystagmus from verticals of either ear; there was correct past pointing after stimulation

of the right ear, but faulty after stimulation of the left ear. There was good nystagmus from the horizontals, with a tendency to conjugate deviation.

On turning to the right, there were subnormal nystagmus and vertigo; on turning to the left, the nystagmus was exaggerated and vertigo was subnormal.

It was suggested that the vestibular examination pointed to a lesion near the pons on the left. The vertigo improved within a few days, although the numbness of the face extended up to the hair margin. Within ten days all symptoms improved and eventually disappeared.

Another examination, one month later (Fig. 9) revealed that all spontaneous signs had disappeared except that the left hand past pointed 2 inches (5 cm.) to the right. Turning revealed normal nystagmus and vertigo responses. The verticals of both sides still failed to yield nystagmus on douching. The horizontals reacted with normal nystagmus. The past pointing was still erratic after stimulation of the left ear. In view of the rapid improvement, it seemed probable that a later examination would reveal normal findings.

The entire examination was made at one time, and after completion of the caloric test the patient became nauseated and vomited.

Case 8.—V. M., aged 14 years, whose chief complaint was deafness, was a healthy looking child and well nourished. The mother was in fair health, except that she was tuberculous and slightly deaf. The grandmother and several uncles were deaf. The patient had a past history of measles, german measles and whooping cough at 5 years; earaches in infancy; headaches quite often in the past few months, mostly frontal. Six years previously she had had a cough, which lasted two years. The tonsils had been removed. There was dizziness at times and tinnitus in the right ear. She had been deaf for four or five years, the right ear being more affected.

Examination, August, 1920, revealed that the nose and throat were negative except that the voice was husky and the cords seemed flabby. Examination of the ears revealed a slight retraction of the right membrana tympani. The left

membrana tympani was pink. The right tube was narrow. The hearing was:

	0	1"	poor	ac < bc > n
C ⁶⁴	—	C —	C ⁴ —	—
	1"	3"	poor	ac < bc > n
	Limit			6"
Galton	—		W. V.	—
	Limit			18"

Weber to right.

The blood Wassermann was negative.

Vestibular examination (entire test at one sitting) (Fig. 10) revealed subnormal nystagmus, vertigo and past pointing; no responses from verticals or horizontals after douching right ear, no responses from verticals of the left ear; good nystagmus from left horizontal but reversed past pointing. Three days later, the douching was repeated with identical results.

The picture was that of a right angle lesion except that there was some hearing in the right ear, but it is poor in both ears.

Eighteen months later reexamination in two stages (Fig. 11) revealed a slight increase in deafness for high tones; nystagmus after turning subnormal and vertigo responses very much diminished; past pointing to right on turning in both directions.

The striking change was apparent on douching. Whereas at the first examination no nystagmus was elicited from any canal except the left horizontal, we now got responses from all canals, although, to be sure, the reaction from the verticals was poor. On turning with the head back 60 degrees there was rotary nystagmus of good amplitude and duration.

The interpretation of these findings was very difficult. While the findings at the first test pointed strongly to some lesion growing toward the angle on the right side, the findings in the last examination seemed to upset this hypothesis completely.

The difficult question to determine was, "Has this patient an intracranial lesion?" The results of the last vestibular test, considered in connection with the high degree of deafness in both ears, suggested a bilateral peripheral lesion. This case

also illustrates the advantage of reexaminations. It is tentatively included in the group of intracranial lesions, but will be studied further.

Case 9.—Dr. J. W. H., aged 37, sustained a basal fracture in an accident five months previously. He was unconscious for three days, bleeding from both ears. When consciousness returned, he was dizzy on sitting up or turning the head to the right. There was right facial paralysis.

All symptoms were now much improved. The patient was dizzy, and there was deafness in the right ear. The right facial muscles were still weak. The membrana tympani was thickened. Hearing was poor for the high tones in the right ear; fair in the left ear.

On vestibular examination, at one sitting (Fig. 12), there was spontaneous crossed past pointing; Romberg sign; little swaying. Poor nystagmus and vertigo were noted after turning. The patient past pointed correctly on turning to the right but faulted with both hands after turning to left. Falling was correct after turning in each direction with the head 120 degrees forward.

On douching, there were no responses from the right ear, and none from the verticals of the left ear, but there were good nystagmus and vertigo from the left horizontal; past pointing was the same as spontaneous.

The picture is similar to that of an angle lesion. The man had either an injury to both labyrinths or it is possible there was a clot in the right angle.

Case 10.—Mr. W. L., aged 22, referred, Feb. 7, 1922, by a neurologist, on account of failing vision, vertigo and convulsions, gave a history of convulsions for eighteen months which were described as follows: face flushed, fingers grip and he falls if standing; becomes unconscious "but can keep talking"; no rigidity nor jerking but if he takes hold of bystander he hangs on; no frothing nor tongue biting. The patient said that his eyes had been crossed for six months, although he was told a year previously that they were not straight. He had headaches in occiput close to axis, and when the pain became severe he had a convulsive attack and sometimes vomited. The head was flexed with vertex to the right and chin to the left. He walked fairly well, though at times, he said,

"It seems as if my feet wanted to go on ahead of me." He had not been able to read for six months. Vision in the right eye was poorer than in the left. He staggered sometimes; he deviated to the left on walking, and sometimes fell to the left and backward.

Neurologic examination revealed marked divergent strabismus, right eye especially being involved. The pupils reacted to light but not to accommodation; there were bilateral choked discs. The patient was able to count fingers at 12 inches (30 cm.) with the left eye, but was not able to count fingers at 4 inches (10 cm.) with the right eye. There was contraction of the upper visual field in left eye. Fields in right eye could not be taken on account of blindness. The eyes could not be raised above horizontal. The tongue protruded in midline without tremor. All other cranial nerves were negative. The reflexes were not significant. There was no adiadokokinesis. Finger to nose and heel to knee movements were normal. Occasionally there were sudden jerking movements of the shoulders, more marked on the left side. Romberg sign was negative. Percussion of the skull was negative, except for tenderness over occiput.

The patient denied venereal disease. The blood Wassermann reaction was + + + +. Blood examination revealed: Hemoglobin, 90 per cent; red blood cells, 5 million; polymorphonuclears, 65 per cent. The urine was negative. Examination of the spinal fluid revealed: Seven lymphocytes per cubic millimeter; globulin positive; no pus cells; Wassermann reaction + + + +.

Vestibular examination (Fig. 13), entire test at one sitting, revealed: No spontaneous nystagmus nor past pointing; Romberg sign: falls to left and backward in all positions of head. Turning produced exaggerated nystagmus and vertigo. On douching there was no nystagmus nor vertigo from right verticals but correct past pointing. There was good but delayed nystagmus from left verticals, with vertigo and correct past pointing, and good responses from both horizontals. No nausea, pallor or sweating was noted.

The hearing was good and drum membranes were intact.

A report was made that the neurootologic tests pointed to a

supratentorial lesion on the right side, probably back of the frontal area.

The exaggeration of the nystagmus and vertigo with good past pointing seemed to rule out the posterior fossa. The fact that the right vertical showed lack of nystagmus and vertigo responses but did show good past pointing, while the horizontals and left verticals gave correct responses, indicated a right sided lesion near the brain stem. The phenomenon that the test was completed at one sitting without producing nausea or vomiting was suggestive of a posterior fossa lesion, but this was thought to be outweighed by the other findings.

The patient came in five weeks later with a history of no improvement and, in fact, vision was still further impaired. Antisyphilitic treatment had been instituted and was being pushed.

Another vestibular test (Fig. 14) yielded similar results: Spontaneous past pointing to right with left hand; negative Romberg sign. The nystagmus time was still further exaggerated; the right verticals still failed to show nystagmus or vertigo, but now, after douching, the nystagmus from the left vertical canals was perverted, there being a rotary nystagmus to the right in the left eye and a nystagmus downward obliquely and to the right in the right eye. The horizontals reacted well, but there was a tendency to conjugate deviation to the left after douching both ears.

A roentgenogram of the skull (Figs. 15 and 16) showed an enlarged sella, with erosion in the floor and a shadow in the sphenoid sinus, although no symptoms of pituitarism had been elicited. In spite of the latter and in view of his rapidly failing vision, it was thought that possibly a tumor was present in the sella and sphenoid and that a transsphenoid operation might give some relief to his blindness and afford time for a more intensive antisyphilitic treatment. During the operation, however, and before the sphenoid sinuses were entered, the patient suddenly had a hard convulsion and died on the table.

Necropsy revealed: Brain removed intact; convolutions flattened over the right hemisphere; definite sensation of resistance felt in the anterior part of the longitudinal fissure.

On section (Figs. 17 and 18), a tumor $1\frac{7}{8}$ by $1\frac{3}{8}$ by $2\frac{1}{2}$ inches (4.7 by 3.4 by 6.2 cm.) soft and reddish in color was

found. It was apparently attached to the floor of the third ventricle and extended upward into the lateral ventricles rather more toward the right side, reaching up to the corpus callosum. There was no direct pressure on the anterior end of the aqueduct of Sylvius. There was pressure on both optic tracts. The lateral ventricles were almost completely filled in their anterior part by the tumor. All structures in the floor of the third ventricle and in the anterior part of the partition between the two lateral ventricles were involved by the pressure.

The sella turcica was flattened on the right side. The floor of the sella was necrotic, and the hypophysis was found in the sphenoid sinus.

Microscopic examination revealed: Numerous polygonal cells with round and oval nuclei, which take stain deeply, growing in solid masses. Occasional islands of degenerated cerebral tissue were found among the cell masses. The diagnosis was glioma cerebri.

301 FERGUSON BUILDING.

REFERENCES.

1. Vail, H. H.: *Laryngoscope* 30:505 (Aug.) 1920.
2. Lyons, H. R.: *Ann. Otol. Rhinol. and Laryngol.* 29:898 (Dec.) 1920.
3. Since this was written, I have learned that Ingham and Jones reported a case at the American Otological Society meeting.

Chart 1
SPONTANEOUS

NYSTAGMUS		POINTING	
		RIGHT	LEFT
Looking to RIGHT	0		
Looking to LEFT	0		
Looking UP	0		
Looking DOWN	0		

Shoulder from above

Nystagmus

Vertigo

Past-pointing

Falling

Romberg

Turning head to right

Turning head to left

Attempt to overthrow

Negative

To RIGHT →	To RIGHT		
Amp. Good	Shoulder from above	24" to R	8" to R
Duration 30 Sec.		8 R	2 R
	Nystagmus	2 R	T
	Vertigo 15 sec.	T	
	Past-pointing Correct		
To LEFT ←	To LEFT		
Amp. GOOD	Shoulder from above	6" to L	6" to L
Duration 31 Sec.		T	2 L
	Nystagmus	T	T
	Vertigo 15 sec		
	Past-pointing Correct		

Douche RIGHT	0	Douche RIGHT		
Amp.		Shoulder from above	4	to R 2 to R
After 3 min.43 sec.				
	Nystagmus 0			
	Vertigo Present			
	Past-pointing Correct			
	Falling			
Head Back →			4	to R 6 to R
Amp. Tremendous				
Douche LEFT ↶		Douche LEFT		
Amp. POOR		Shoulder from above	2	to L 2 to L
After 1 min.35 sec.				
	Nystagmus Barely perceptible			
	Vertigo Present			
	Past-pointing Correct			
	Falling			
Head Back ←			2	to L 2 to L
Amp. Wide				
Right eye tends to turn inward.				
Entire test at one sitting--no nausea, pallor or sweat.				

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Fig. 1 (Case 1).—Results of vestibular examination.

Chart 2

Mrs. E.R.W.

TESTS OF THE VESTIBULAR APPARATUS

NYSTAGMUS		SPONTANEOUS		POINTING	
				RIGHT	LEFT
Looking to RIGHT	None	Shoulder from above		T	T
Looking to LEFT	None				
Looking UP	None	Nystagmus	None	Hearing: C 64 3" 6" 4 good limit 2" C 6" 4 good Galt limit W.V. 20' 20' Weber * ac > bc < n ac > bc < n	
Looking DOWN	None	Vertigo	Yes		
		Past-pointing	Falling		
		Romberg	Very slight swaying		
		Turning head to right			
		Turning head to left			
		Attempt to overthrow			
		TURNING			
To RIGHT →		To RIGHT			
Amp. Small		Shoulder from above		12 to R	2 to R
Duration 17 Sec.				2 R	2 R
		Nystagmus	Subnormal	T	8 R
		Vertigo	5 sec.		8 R
		Past-pointing	Exaggerated with L.H.		8 R
To LEFT ←		To LEFT			
Amp. Small		Shoulder from above		8 to L	6 to L
Duration 18 Sec.				T	T
		Nystagmus	Subnormal		
		Vertigo	5 sec.		
		Past-pointing	Subnormal		
		CALORIC			
Douche RIGHT	None	Douche RIGHT			
Amp.		Shoulder from above		T to	6 to R
After 2 min. 45 sec.					
		Nystagmus	None		
		Vertigo	Slight		
		Past-pointing	Fault with R.H.		
		Falling			
Head Back →		Slight nausea		2 to R	6 to R
Amp. Fair.					
Douche LEFT ↶		Douche LEFT			
Amp. Small		Shoulder from above		to L	to L
After 1 min. 37 sec.					
		Nystagmus	Delayed		
		Vertigo	Marked		
		Past-pointing	Correct		
		Falling			
Head Back ←		Quite nauseated		1 to L	T to
Amp. Good.					

Fig 2 (Case 2).—Results of vestibular examination.

Chart 3

H.K.-16yrs.

TESTS OF THE VESTIBULAR APPARATUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

SPONTANEOUS

Shoulder from above
Nystagmus None
Vertigo None
Past-pointing With L.H.
Falling
Romberg
Turning head to right
Turning head to left
Attempt to overthrow

POINTING

RIGHT LEFT
T 1 to L

Hearing: 64-3-12-4 good Salt-
4-12- good
W.V.-----Webber-----
as < bc > as
bc > cc = n

TURNING

To RIGHT →
Amp. Small
Duration 16 Sec.

To RIGHT
Shoulder from above
Nystagmus Subnormal
Vertigo 4 sec.
Past-pointing Fault with L.H.

20 to R 2 to
4 R 1 L
2 R 2 L
1 1

To LEFT ←
Amp. Good
Duration 24 Sec.

To LEFT
Shoulder from above
Nystagmus Good
Vertigo 10 sec.
Past-pointing Correct

20 to L 1 to L
4 L 1 L
1 1

CALORIC

Douche RIGHT None
Amp. Small
After 5 min. sec.

Douche RIGHT
Shoulder from above
Nystagmus None
Vertigo (Nausea, sweat and pallor.)
Past-pointing None
Falling

T to 2 to L

Head Back →
Amp. Good

T to 4 to 5

Douche LEFT ←
Amp. Small
After 2 min. 30 sec

Douche LEFT
Shoulder from above
Nystagmus Barely perceptible
Vertigo yes.
Past-pointing Questionable
Falling

2 to L 1 to L

Head Back ←
Amp. Good

T to T to

Entire test at one sitting. Pallor, nausea, sweat. Head dropped forward and was apparently stupid. Tendency to fall forward.

From Dr. I. H. Jones' Equilibrium and Vertigo.

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Fig. 3 (Case 3).—Results of vestibular examination.

TESTS OF THE VESTIBULAR APPARATUS

Chart 4 SPONTANEOUS

Mrs. McC.

NYSTAGMUS

Looking to RIGHT	0	Shoulder from above
Looking to LEFT	0	
Looking UP	0	Nystagmus
Looking DOWN	0	Vertigo Present
		Past-pointing
		Falling Tends to fall to left
		Romberg Slight swaying
		Turning head to right same
		Turning head to left same
		Attempt to overthrow

POINTING RIGHT LEFT

T T

TURNING

To RIGHT →
Amp. Good
Duration 30 Sec.

To RIGHT
Shoulder from above
Nystagmus
Vertigo 23 sec
Past-pointing Correct

12 to R	15 to R
6 R	6 R
3 R	1 R
2 R	T
T	

To LEFT ←
Amp. Good
Duration 30 Sec.

To LEFT
Shoulder from above
Nystagmus
Vertigo 19 sec
Past-pointing Correct.

12 to L	12 to L
T	3 L
T	2 L
	2 L
	T

CALORIC

Douche RIGHT 0
Amp.
After 3 min. 10 sec.

Douche RIGHT
Shoulder from above
Nystagmus
Vertigo slight
Past-pointing Fault with R.H.
Falling

T to 6 to R

Head Back →
Amp. small, slow

T to 6 to R

Douche LEFT ↙
Amp. almost none
After 1 min. 24 sec.

Douche LEFT
Shoulder from above
Nystagmus poor reaction
Vertigo slight
Past-pointing None
Falling

T to T to

Head Back ←
Amp. GOOD

T to T to

Examined in two sittings. Vomited after getting home.

Fig. 4 (Case 4).—Results of vestibular examination.

TESTS OF THE VESTIBULAR APPARATUS

Chart 5 SPONTANEOUS

A.P.M.

NYSTAGMUS

POINTING

RIGHT LEFT
 $\frac{1}{2}$ to R T
T 3 to R

Looking to RIGHT 0 Shoulder from above
Looking to LEFT 0
Looking UP 0 Nystagmus
Looking DOWN 0 Vertigo
Past-pointing
Falling
Romberg **Negative**
Turning head to right
Turning head to left
Attempt to overthrow

TURNING

To RIGHT → Amp. **Good**
Duration 17 Sec. Shoulder from above
Nystagmus
Vertigo 17 sec.
Past-pointing **Correct**

4 to R 4 to R
1 R 4 R
T 3 R
T

To LEFT ← Amp. **Good**
Duration 25 Sec. Shoulder from above
Nystagmus
Vertigo 23 sec.
Past-pointing **Correct**

6 to L 6 to L
2 L 2 L
2 R 2 R

CALORIC

Douche RIGHT 0 Amp.
After 3 min. 55 sec. Douche RIGHT
Shoulder from above

T to T to

Nystagmus
Vertigo **None**
Past-pointing **None**
Falling

Head Back → Amp. **Good**

Dizzy

T to 2 to R

Douche LEFT 0 Amp.
After 4 min 23 sec. Douche LEFT
Shoulder from above

2 to L 1 to R

Nystagmus
Vertigo **None**
Past-pointing **Crossed**
Falling

Head Back ← Amp. **Wide**

Slight vertigo.

2 to L 1 to R

Examined in two sittings.

Fig. 5 (Case 5).—Results of vestibular examination.

TESTS OF THE VESTIBULAR APPARATUS

Chart 6

SPONTANEOUS

Mrs. E. T. P.

NYSTAGMUS

POINTING RIGHT LEFT

Looking to RIGHT 0	Shoulder from above		
Looking to LEFT 0		T	T
Looking UP 0	Nystagmus		
Looking DOWN 0	Vertigo		
	Past-pointing		
	Falling		
	Romberg Falls backward		
	Turning head to right ditto		
	Turning head to left ditto		
	Attempt to overthrow		

TURNING

To RIGHT → Amp Small Duration 16 Sec.	To RIGHT Shoulder from above	2 to R 2 R T	4 to R 4 R 2 R 2 R T
	Nystagmus Subnormal		
	Vertigo 14 sec		
	Past-pointing Correct		
To LEFT ← Amp Small Duration 17 Sec.	To LEFT Shoulder from above	1 to L 2 L 2 L 2 L	T to T T T
	Nystagmus Subnormal		
	Vertigo 22 sec.		
	Past-pointing Fault, L.H.		

CALORIC

Douche RIGHT 0 Amp After 5 min. sec.	Douche RIGHT Shoulder from above	1/2 to L	T to
	Nystagmus None		
	Vertigo None		
	Past-pointing None		
	Falling to right		
Head Back → Amp very fine	Dizzy.	T to	3 to R
Douche LEFT Amp POOR After 5 min. sec.	Douche LEFT Shoulder from above	1/2 to L	T to
	Nystagmus Barely a trace		
	Vertigo None		
	Past-pointing None		
	Falling slightly to right		
Head Back ↖ Amp Fair	Slightly dizzy	1/2 to L	T to

Test in two sittings. No nausea, pallor or sweat.

From Dr. I. H. Jones' Equilibrium and Vertigo.

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Fig. 6 (Case 6).—Results of first vestibular examination.

Mrs. E. T. P.

TESTS OF THE VESTIBULAR APPARATUS
Chart 7
SPONTANEOUS

NYSTAGMUS

Looking to RIGHT 0
Looking to LEFT 0
Looking UP 0
Looking DOWN 0

Shoulder from above
Nystagmus
Vertigo
Past-pointing
Falling
Romberg Sways to right & back.
Turning head to right . same
Turning head to left steady
Attempt to overthrow

POINTING
RIGHT T LEFT T

To RIGHT →
Amp. Fair
Duration 19Sec.

TURNING
To RIGHT
Shoulder from above
Nystagmus
Vertigo 23 sec.
Past-pointing Correct

4 to R
T
6 to R
2 R
2 R
T

To LEFT ←
Amp. Small
Duration 19Sec.

To LEFT
Shoulder from above
Nystagmus
Vertigo 23 sec.
Past-pointing

T to T to

CALORIC

Douche RIGHT ↷
Amp. POOR
After 3 min. 4 sec

Douche RIGHT
Shoulder from above
Nystagmus Did not improve
Vertigo None
Past-pointing None
Falling

T to T to

Head Back →
Amp. Good.

Dizzy

1 to R 1 to R

Douche LEFT ↶
Amp. Very poor
After 2 min. 25sec.

Douche LEFT
Shoulder from above
Nystagmus Did not improve
Vertigo None
Past-pointing Fault, L.R.
Falling

1 to L T to

Head Back ↖
Amp. Fair

1 to L 1 to L

Entire test at one sitting--no nausea, pallor or sweat.

Fig. 7 (Case 6).—Results of second vestibular examination.

Miss M. S.

TESTS OF THE VESTIBULAR APPARATUS
Chart 8
SPONTANEOUS

NYSTAGMUS		POINTING	
		RIGHT T	LEFT 4 "to L
Looking to RIGHT ←	Shoulder from above		
Looking to LEFT →			
Looking UP ↑ ↗	Nystagmus Vertigo Past-pointing Falling		
Looking DOWN 0	Romberg Falls to left Turning head to right Falls to left Turning head to left Falls backward Attempt to overthrow		
TURNING			
To RIGHT →	To RIGHT Shoulder from above	to	to
Amp. Poor	Nystagmus Subnormal		
Duration 11Sec.	Vertigo 13 sec		
	Past-pointing Not taken		
To LEFT ←	To LEFT Shoulder from above	to	to
Amp. Good	Nystagmus		
Duration 41Sec.	Vertigo 11 sec		
	Past-pointing Not taken		
CALORIC			
Douche RIGHT 0	Douche RIGHT Shoulder from above	4 to R	4 to R
Amp.	Nystagmus		
After 4 min. sec.	Vertigo		
	Past-pointing		
	Falling		
Head Back →		10 to R	10 to R
Amp. Wide; tendency to conjugate deviation			
Douche LEFT 0	Douche LEFT Shoulder from above	T to	4 to L
Amp.	Nystagmus		
After 3 min. 55 sec.	Vertigo		
	Past-pointing		
	Falling		
Head Back ←		T to	4 to R
Amp. Small; tendency to conjugate deviation.			

Test in two sittings.

Fig. 8 (Case 7).—Results of first vestibular examination.

TESTS OF THE VESTIBULAR APPARATUS
Chart 9
SPONTANEOUS

Miss M.G.

NYSTAGMUS

Looking to RIGHT	0	Shoulder from above
Looking to LEFT	0	
Looking UP	0	Nystagmus 0
Looking DOWN	0	Vertigo 0
		Past-pointing With L.H.
		Falling
		Romberg Negative
		Turning head to right
		Turning head to left
		Attempt to overthrow

POINTING
RIGHT T LEFT 2 to R

TURNING

To RIGHT →		To RIGHT		
Amp. Good		Shoulder from above	to	to
Duration 26 Sec.				
		Nystagmus Normal		
		Vertigo 23 sec.		
		Past-pointing Not taken		
To LEFT ←		To LEFT		
Amp. Good		Shoulder from above	to	to
Duration 31 Sec.				
		Nystagmus		
		Vertigo 24 sec.		
		Past-pointing Not taken		

CALORIC

Douche RIGHT 0		Douche RIGHT		
Amp.		Shoulder from above	T to	2 to R
After 3 min 35 sec.				
		Nystagmus None		
		Vertigo Slight		
		Past-pointing Fault, R.H.		
		Falling To right	½ to R	4 to R
Head Back →		Dizzy--nauseated		
Amp. Good				
Douche LEFT 0		Douche LEFT		
Amp.		Shoulder from above	T to	2 to R
After 1 min. 55 sec.				
		Nystagmus None		
		Vertigo Began at 1 min.		
		Past-pointing Fault, both hands		
		Falling	3 to L	2 to R
Head Back ←				
Amp. Good				

Entire examination at one sitting.

Fig. 9 (Case 7).—Results of second vestibular examination.

Chart 10

J. M. Age 14

TESTS OF THE VESTIBULAR APPARATUS

NYSTAGMUS		SPONTANEOUS		POINTING	
				RIGHT	LEFT
Looking to RIGHT	None	Shoulder from above		T	T
Looking to LEFT	None	Nystagmus None			
Looking UP	None	Vertigo Yes			
Looking DOWN	None	Past-pointing None			
		Falling			
		Romberg No swaying			
		Turning head to right			
		Turning head to left			
		Attempt to overthrow	Pelvic movements normal		
Hearing: C64 0 1" 4 0 Salt 2.3 1 3" poor 0.3 W.V. 6" Weber * ac < bc > n 18" ac < bc > n					
TURNING					
To RIGHT →		To RIGHT			
Amp. Fair-slow		Shoulder from above		2" to R	T to
Duration 13 Sec.		Nystagmus subnormal			
		Vertigo 9 sec.			
		Past-pointing Fault			
To LEFT ←		To LEFT			
Amp. Fair-slow		Shoulder from above		T to	T to
Duration 13 Sec.		Nystagmus subnormal			
		Vertigo 10 sec.			
		Past-pointing None			
CALORIC					
Douche RIGHT	None	Douche RIGHT			
Amp.		Shoulder from above		T to	T to
After 4 min. 5 sec.		Nystagmus None			
		Vertigo None			
		Past-pointing None			
		Falling			
Head Back	None			T to	T to
Amp.					
Douche LEFT	None	Douche LEFT			
Amp.		Shoulder from above		T to	T to
After 3 min. 6 sec.		Nystagmus None			
		Vertigo None			
		Past-pointing None			
		Falling			
Head Back ←				8" to R	8" to R
Amp. Good-slow					

Entire test at one sitting; slight nausea--did not vomit.

From Dr. I. H. Jones' Equilibrium and Vertigo.

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Fig. 10 (Case 8).—Results of first vestibular examination.

Chart 11

V.M.

TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS		POINTING									
NYSTAGMUS		RIGHT	LEFT								
Looking to RIGHT None	Shoulder from above	T	T								
Looking to LEFT None											
Looking UP None	Nystagmus None	<table><tr><td>Turning, head back 60°</td><td></td></tr><tr><td>To right ↶</td><td>To left ↷</td></tr><tr><td>Amp. Fair</td><td>Amp. Good</td></tr><tr><td>Duration 14 sec</td><td>Duration 15 sec</td></tr></table>		Turning, head back 60°		To right ↶	To left ↷	Amp. Fair	Amp. Good	Duration 14 sec	Duration 15 sec
Turning, head back 60°											
To right ↶	To left ↷										
Amp. Fair	Amp. Good										
Duration 14 sec	Duration 15 sec										
Looking DOWN None	Vertigo Very little										
	Past-pointing None										
	Falling										
	Romberg No swaying										
	Turning head to right										
	Turning head to left										
	Attempt to overthrow										
TURNING											
To RIGHT →	To RIGHT										
Amp. Fair	Shoulder from above	4 to R	2 to R								
Duration 16 Sec.		3 R	2 R								
	Nystagmus Subnormal	2 R	2 R								
	Vertigo 4 sec.	T	T								
	Past-pointing Correct	.									
To LEFT ←	To LEFT										
Amp. Good	Shoulder from above	2 to R	4 to R								
Duration 18 Sec.		T	2 R								
	Nystagmus Subnormal		T								
	Vertigo 5 sec.										
	Past-pointing Reversed										
CALORIC											
Douche RIGHT ↷	Douche RIGHT										
Amp. Small-poor	Shoulder from above	1 to R	T to								
After 3 min. 55 sec.											
	Nystagmus Poor										
	Vertigo Very little										
	Past-pointing Fault with L.H.										
	Falling										
Head Back →	Slight vertigo	r. 4 to R	4 to R								
Amp. Good											
Douche LEFT ↶	Douche LEFT										
Amp. Small-poor	Shoulder from above	1 to R	1 to L								
After 2 min. 10 sec.											
	Nystagmus Poor										
	Vertigo None										
	Past-pointing Reversed with R.H.										
	Falling										
Head Back ←	Slight vertigo.	1 to L	1 to L								
Amp. Good											

Test in two sittings; nauseated but did not vomit

From Dr. I. H. Jones' Equilibrium and Vertigo.

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Fig. 11 (Case 8).—Results of second vestibular examination.

Chart 12

Dr. J. W. H.

TESTS OF THE VESTIBULAR APPARATUS

NYSTAGMUS		SPONTANEOUS		POINTING	
				RIGHT	LEFT
Looking to RIGHT	None	Shoulder from above		2 to L	2 to R
Looking to LEFT	None				
Looking UP	None	Nystagmus None			
Looking DOWN	None	Vertigo Yes			
		Past-pointing Crossed			
		Falling			
		Romberg Negative			
		Turning head to right			
		Turning head to left			
		Attempt to overthrow			
		Deviates to right on walking with closed eyes			
TURNING					
To RIGHT →		To RIGHT			
Amp. Small		Shoulder from above		21 to R	6 to R
Duration 9 Sec.				2 R	2 R
		Nystagmus Subnormal		2 R	2 R
		Vertigo 11 sec.		T	T
		Past-pointing Correct			
		Falls to right--slight.			
To LEFT ←		To LEFT			
Amp. Small		Shoulder from above		T to	4 to R
Duration 4 Sec.				T	2 R
		Nystagmus Subnormal		T	2 R
		Vertigo 3 sec.			T
		Past-pointing Reversed with L.H.			
		Falls to left--marked.			
CALORIC					
Douche RIGHT	None	Douche RIGHT			
Amp.		Shoulder from above		2 to L	2 to R
After 5 min. sec.					
		Nystagmus None			
		Vertigo None			
		Past-pointing Same as spontaneous			
		Falling To right			
Head Back	None			2 to L	2 to R
Amp.					
Douche LEFT	None	Douche LEFT			
Amp.		Shoulder from above		2 to L	2 to R
After 5 min. sec.					
		Nystagmus None			
		Vertigo None			
		Past-pointing Same as spontaneous			
		Falling			
Head Back ←				2 to L	2 to R
Amp. Good		Is dizzy			

Hearing: C ₄₄	3"	6"	10"	15"	20"	25"	30"	35"	40"	45"	50"	55"	60"	65"	70"	75"	80"	85"	90"	95"	100"
W.V.	18"	15"	12"	10"	8"	6"	4"	3"	2"	1"	0"	0"	0"	0"	0"	0"	0"	0"	0"	0"	0"
W.V.	18"	15"	12"	10"	8"	6"	4"	3"	2"	1"	0"	0"	0"	0"	0"	0"	0"	0"	0"	0"	0"

Entire test at one sitting. No nausea, pallor or sweat.

From Dr. J. H. Jones' Equilibrium and Vertigo.

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Fig. 12 (Case 9).—Results of vestibular examination.

TESTS OF THE VESTIBULAR APPARATUS
W.L.-Chart 13
SPONTANEOUS

NYSTAGMUS		POINTING	
		RIGHT	LEFT
Looking to RIGHT	0	T	T
Looking to LEFT	0		
Looking UP	0		
Looking DOWN	0		
	Nystagmus Vertigo Past-pointing Falling Romberg Turning head to right Turning head to left Attempt to overthrow		

Falls to left and back

TURNING			
To RIGHT →	To RIGHT		
Amp. Wide	Shoulder from above	to	to
Duration 43 Sec.			
	Nystagmus Vertigo 33 sec Past-pointing		
To LEFT ←	To LEFT		
Amp. Wide	Shoulder from above	to	to
Duration 34 Sec.			
	Nystagmus Vertigo 31 sec. Past-pointing		

CALORIC			
Douche RIGHT	Douche RIGHT		
Amp. 0	Shoulder from above	6" to R	6" to R
After 5 min. sec.			
	Nystagmus None Vertigo None Past-pointing Correct Falling To right		
Head Back →	Vertigo	6" to R	12" to R
Amp. Wide			
Douche LEFT ↙	Douche LEFT		
Amp. Good	Shoulder from above	4" to L	6" to L
After 1 min. 50 sec.			
	Nystagmus Delayed Vertigo Present Past-pointing Correct Falling To left		
Head Back ←	Vertigo marked	8" to L	11" to L
Amp. Wide			
Tendency to conjugate deviation.			
	Entire test at one sitting.		

From Dr. I. H. Jones' Equilibrium and Vertigo.

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Fig. 13 (Case 10)—Results of first vestibular examination.

TESTS OF THE VESTIBULAR APPARATUS

Chart 14

SPONTANEOUS

W.L. 22

NYSTAGMUS		POINTING	
Looking to RIGHT 0	Shoulder from above	RIGHT T	LEFT 2" to R
Looking to LEFT 0	Nystagmus		
Looking UP 0	Vertigo		
Looking DOWN 0	Past-pointing		
	Falling		
	Romberg Negative		
	Turning head to right		
	Turning head to left		
	Attempt to overthrow		
TURNING			
To RIGHT → Amp. Wide Duration 60 Sec.	To RIGHT Shoulder from above	to	to
	Nystagmus Exaggerated		
	Vertigo 24 sec.		
	Past-pointing		
To LEFT ← Amp. Wide Duration 37 Sec.	To LEFT Shoulder from above	to	to
	Nystagmus		
	Vertigo 34 sec		
	Past-pointing		
CALORIC			
Douche RIGHT 0 Amp. After 5 min. sec.	Douche RIGHT Shoulder from above	6" to R	8" to R
	Nystagmus None		
	Vertigo None		
	Past-pointing Correct		
	Falling		
Head Back → Amp. Wide,	tendency to conjugate deviation	6" to R	14" to R
Douche LEFT ↗ ↘ Amp. Good After 1 min 30 sec.	Douche LEFT Shoulder from above	6" to L	1" to L
↙ in R eye	Nystagmus Perverted		
↘ in L eye	Vertigo None		
	Past-pointing correct		
	Falling		
Head Back ← Amp. Good	No vertigo	6" to L	T to
	Tendency to conjugate deviation		

Entire test at one sitting

From Dr. I. H. Jones' Equilibrium and Vertigo.

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Fig. 14 (Case 10).—Results of second vestibular examination.



Fig. 15 (Case 10).—Location of enlarged sella, showing erosion in the floor and a shadow in the sphenoid sinus.

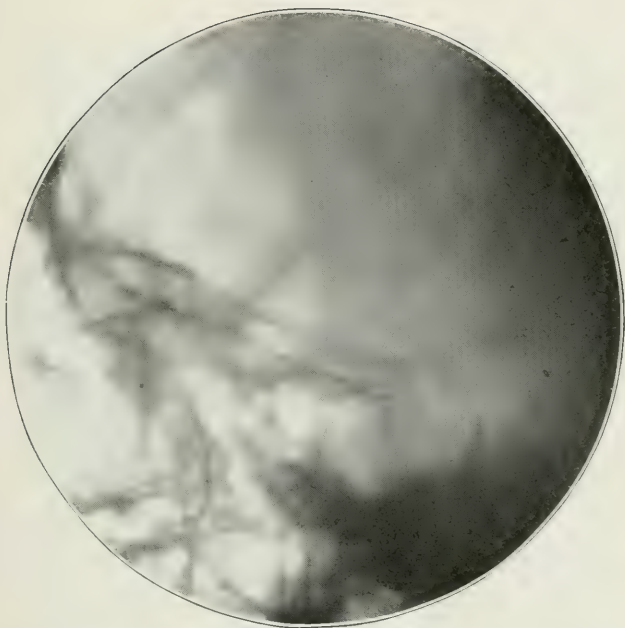


Fig. 16 (Case 10).—Enlarged sella, erosion in floor and shadow in the sphenoid sinus.

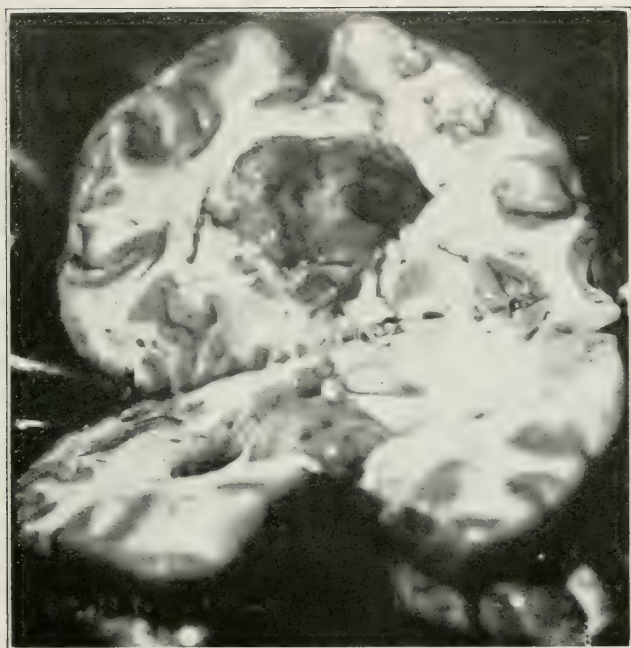


Fig. 17 (Case 10).—Frontal section of brain.



Fig. 18 (Case 10).—Longitudinal section of brain.

XIII.

A SIMPLE KNOT FOR INTRANASAL SUTURES.*

BY WILSON JOHNSTON, M. D.,

PORTLAND, OREGON.

The placing and tying of ligatures and sutures in locations not freely accessible, as in the nose and throat, is always difficult. Anything that tends to remove this difficulty needs no apology for being presented to the profession.

I have used this knot for a number of years and conceived the idea from seeing a surgeon lock a double threaded, continuous suture before tying it. The knot is applicable to all ligatures and sutures placed by a needle on a handle, a needle with the eye in the point, or a needle of the crochet hook type. Whatever the type of needle selected, it must be armed with a double suture and passed through both flaps of the wound. The thread is seized by a hook or a pair of forceps, just back of the eye of the needle (Fig. 1) and held fast while the needle is pushed back along the path of entrance and disengaged from the suture (Fig. II). We now have a loop of suture on one side of the wound and two free ends on the other. Pass one free end through the loop (Fig. III) and make traction on both free ends of the suture. This causes the loop to slip down toward the wound, brings the parts in apposition and draws a tight, secure knot.

In wounds where no tension is expected, this knot is sufficient if the suture is cut so as to leave the ends a quarter to a half inch long. In the majority of wounds it should be reinforced by a loop of the ordinary square knot (Fig. IV). When so reinforced it can be removed, if necessary, by catching one of the free ends with a forcep and making a to and fro motion which causes the reinforcing loop to untie and the suture slips on gentle traction.

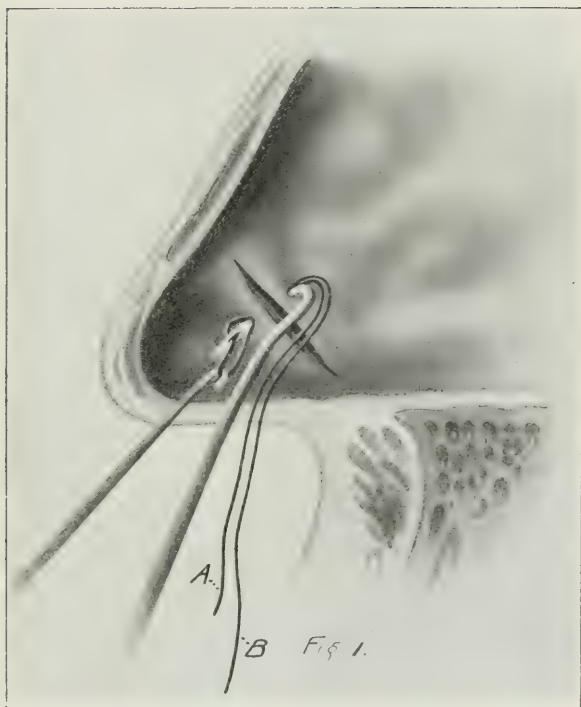
*Read before the Pacific Coast Oto-Ophthalmologic Society at Salt Lake City, Utah, September 15, 1922.

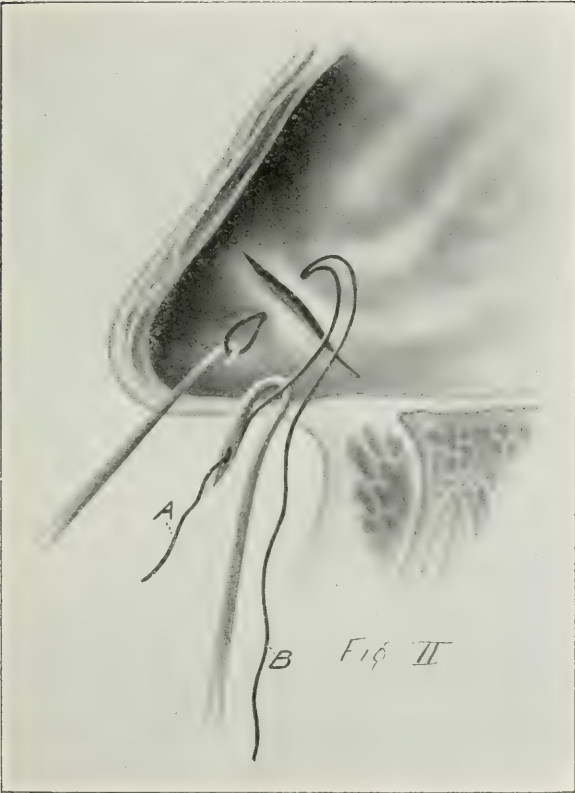
When used to tie ligatures, the knot should be reinforced with several loops of the square knot.

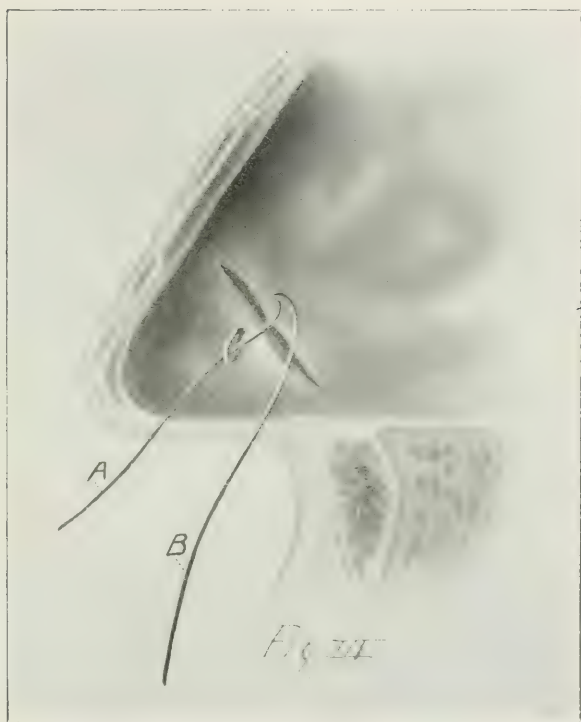
In an emergency, when a needle on a handle is not convenient, an ordinary surgical needle and needle holder may be used. The needle must be threaded with an extra long double suture, which is drawn well through the flap of the wound. The needle is then cut off and the free ends on that side tied, thus reestablishing the loop, and the knot can be tied as described.

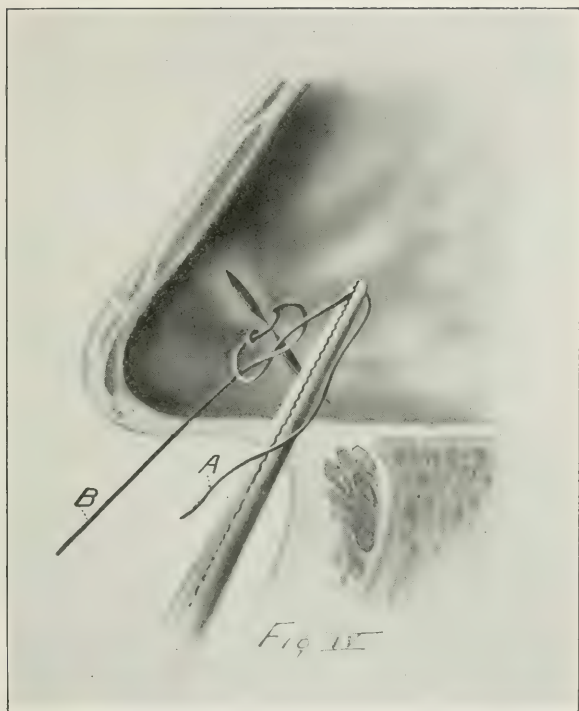
I believe this knot has the following advantages :

1. Is simple.
2. Easily tied.
3. Reliable ; will hold.
4. Requires no special instruments.
5. Easily removed.









XIV.

COMPENSATORY NASAL GROWTHS.*

BY J. A. PRATT, M. D., F. A. C. S.,

INSTRUCTOR OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
MEDICAL SCHOOL, UNIVERSITY OF MINNESOTA,

MINNEAPOLIS.

Since 1910, I have advocated conservation of the turbinates. Up to the present time men are still removing turbinates for nasal stenosis, instead of doing a submucous resection of the septum.

While we can excuse those who read little and never attend medical meetings, we cannot excuse men who pretend to be progressive.

I think I could safely say that at present there are now no nasal surgeons or men who pretend to do nasal surgery, who remove all of the lower turbinate. Those who have, in the past, removed part of the lower turbinate to make a larger opening into the antrum when the trocar washings have not cured the case, are finding they can perform this operation and still retain the lower turbinate.

In the past nasal surgeons have found it necessary to remove a part or all of the middle turbinate in order to exenterate the anterior or posterior ethmoids or open the frontal sinus intranasally.

For many years I have been performing an operation for exenterating the anterior and posterior ethmoids, opening the frontal sinuses or removing the anterior wall of the sphenoid intranasally, without removing any part of the middle turbinate. This operation was first presented to the American Academy at Cleveland in 1919.

*Read before the North Dakota Academy of Ophthalmology and Otolaryngology at Jamestown, North Dakota, June 1, 1922.

For some time I have observed soft meaty growths, of a hyperplastic nature but looking very much like turbinal tissue, growing out from the lateral wall of the nasal cavity or from the septum.

It was not for some months after first noticing these growths that they were associated in my mind with a roomy nose. The position of these growths with the physiologic protection they would give to the nose demonstrate their compensatory character.

These compensatory growths are found on the septum at about the position of the septum tubercle, and they are undoubtedly a hyperplasia of that tissue.

The anterior lateral wall growths are not anterior to the posterior edge of the nasal process of the maxillary bone but begin at the anterior attachment of the middle turbinate, following the anterior edge of the uncinate process, and seem to be a hyperplasia of the soft tissues of the uncinate process.

In this position they protect the frontal sinus opening, the infundibular and bullar regions, which are normally covered by the middle turbinate.

The posterior growths on the lateral wall develop along the posterior half of the attachment of the middle turbinate and protect the posterior ethmoids and sphenoids from irritating air currents.

One of the first cases to attract my attention was a patient, female, age 45, who had entered at the university dispensary. While the membrane of the nose looked healthy and there was no discharge, the nasal cavity was extremely roomy, the turbinates were rudimentary in form, and the nose had the appearance of having had all the turbinates removed.

In the front half of the nasal cavity, beginning at about the anterior attachment of the middle turbinate and extending downward to a little below the attachment of the lower turbinates, was a large, thick, meaty growth, extending nearly to the septum. The growth was present on both sides and re-

sembled, in a crude way, the anterior end of the middle turbinate.

The patient gave a history of a very severe case of atrophic rhinitis, with the formation of enormous crusts all during her girlhood, but now she was free of all nasal trouble.

After satisfying myself that the growths were compensatory, I called one of my fellowship men, with whom I had discussed compensatory growths of the turbinates and ethmoids, and without suggestive assistance he named the growths compensatory.

Since having our attention fixed on these growths we have found them in many cases where the middle turbinates have been removed for exenteration of the ethmoids. What a lesson nature has given us against mutilation of the turbinates. If all other arguments were forgotten, this would prove the case.

In some cases where we have a large nasal space, due to a deflected septum, where the middle turbinate and the ethmoid capsule are compensatorally hypertrophied and we have a suppurating frontal sinus present, I have seen the irritating discharge cause an anterior growth to develop, giving the appearance of a double middle turbinate. Upon examination the growth will be found to spring from the lateral wall, in the position of the anterior compensatory growth, showing they grow from irritation.

The first opportunity I have had to register any definite date as to how long a time it takes for these compensatory growths to develop was afforded me on Saturday, May 6, 1922.

The patient came for an acute exacerbation of a chronic frontal sinusitis. Two years before one of our well known specialists, a man with whom I was in Vienna in 1904, removed the anterior half of her middle turbinate for sinus trouble.

The patient had a perfectly marked compensatory growth, which could easily be confused as the anterior end of a middle turbinate. The growth was allowed to remain in place and

the frontal sinus opened just back of it, in the normal position. This places the development of the growths within two years.

Many men may have noticed these growths and appreciated their significance, but they have not reported them, as far as I know.

With the prevalence, in the past, of the removal of turbinates, I am sure, with a little care and time, we can all see these compensatory growths.

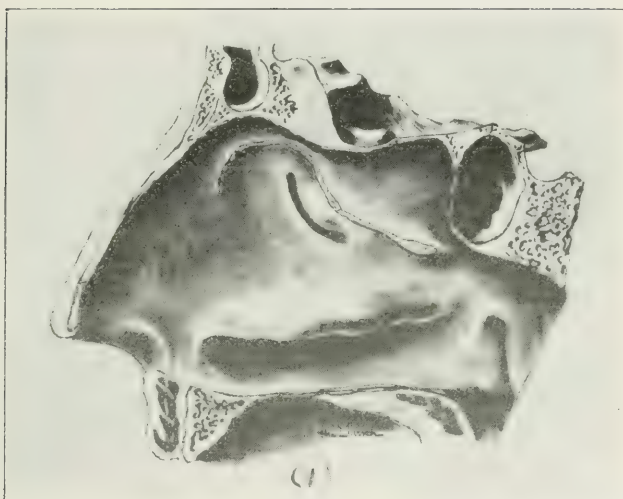


Figure No. 1. Showing a normal lateral wall with the middle turbinate removed. This gives a relative idea of the position of the compensatory growths shown in Figure No. 2.

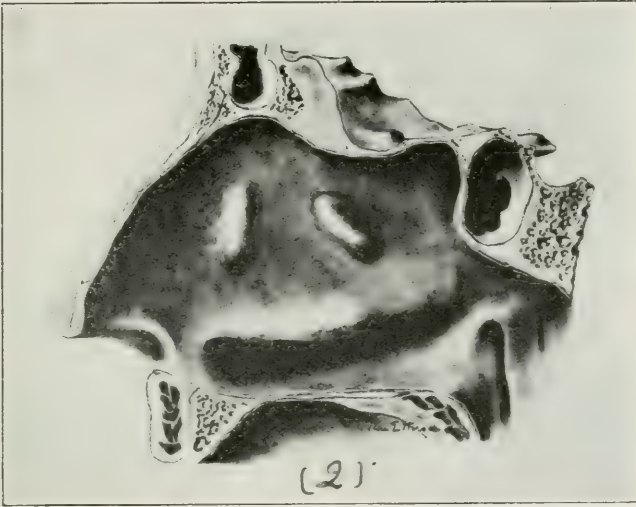


Figure No. 2. Position of the compensatory growth on the lateral wall. Other landmarks of the lateral wall have been effaced to clearly show the position of these growths.

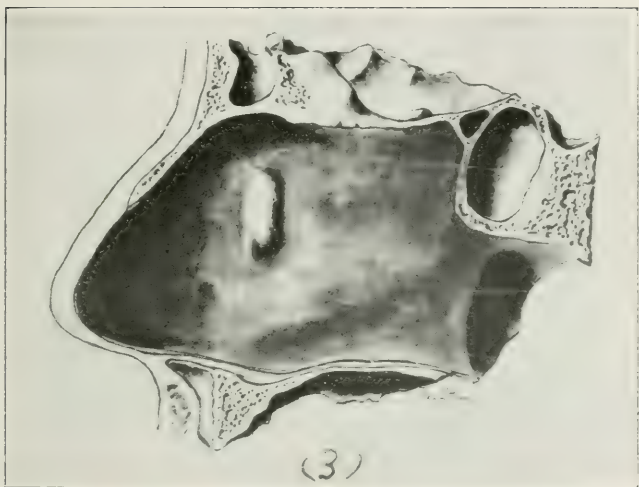


Figure No. 3. The position of the compensatory growth on the septum when it is present.

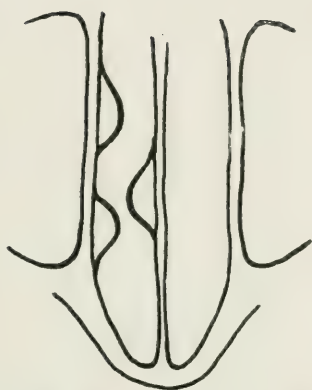


Figure No. 4. A diagrammatic horizontal cut through the nasal chamber showing the relative position of these compensatory growths and how they compensate for the loss of the turbinates.

XV.

REPORT OF A CASE OF TONSILLAR CYST WHICH PROTRUDED FROM THE TONSIL AS A PEDUNCULATED TUMOR.

BY ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

Mrs. X, white, aged about 28, applied at Washington University Dispensary, complaining of a mass in the throat which interfered with swallowing. The mass was of slow growth and was not painful.

Examination showed a pedunculated tumor about the size of the tonsil, lying mesial to it, against the posterior wall of the pharynx. The surface was irregular and somewhat resembled a papilloma. On first examination it appeared to be attached to the posterior pillar, but closer inspection showed the attachment to be at the superior pole of the tonsil. The remainder of the pharynx was negative. The tonsil with the mass attached was removed by Dr. W. M. C. Bryan. The patient made an uneventful recovery.

The specimen on examination shows the tumor mass and the tonsil of equal size and bound together at the superior pole by an attachment 6 mm. in diameter, the tumor having about the same firm consistency as the tonsil. A section was made at right angles to the long axis of the tonsil and tumor, through the pedicle. Much to our surprise the mass proved to be a lacunar retention cyst.

The occluded crypt, which can be traced to the capsule of the tonsil, was firmly sealed by some former acute inflammatory process. As the retained detritus increased in bulk, the tonsil tissue which covered it was forced out and mucous membrane and submucosa proliferated, while the lymph follicles, caught between the wall and the advancing cyst, were all but destroyed.

The tumor was covered with mucous membrane only slightly thicker than normal. The cyst wall was composed of thick

connective tissue in which the follicles were to be found at intervals. The cyst was lined with low columnar epithelium, which in places appeared to undergo pressure atrophy. The wall varied in thickness from .5 mm to 2 mm. Portions of this wall were composed of approximately normal tonsil tissue, while other portions showed local chronic inflammatory changes, thick strata of loose connective tissue and some solid masses of fibrous tissue.

The writer has been unable to discover in the literature any record of tonsil cysts lying without the body of the tonsil. Ballenger, under the heading "Cystoma," dismisses the subject with the statement that cystoma of the tonsil is rare, that it may be either superficial or deep, varies in size, and may contain a quantity of fluid or a mass of inspissated secretions and epithelial debris.

Harry A. Barnes in his *Monograph on the Tonsils* (Mosby, 1914) says: "Cyst of the tonsil results from an inflammatory occlusion of the mouth of the crypt, and the subsequent dilatation of the resultant blind sac by the accumulation of cheesy detritus. It may be situated anywhere in the tonsil, and when small is easily confounded with chronic abscess. Cysts occasionally attain great size. One that I removed seemed to have absorbed the whole tonsil."

Phillips says: "Tonsillar cysts usually result from the inflammatory closure of the lacunar mouths, beneath which collections of caseous matter become encysted. They are also believed to result from traumatism and from the use of the galvanocautery."

This case, while presenting nothing new from the standpoint of pathology, is nevertheless unusual and interesting from a diagnostic angle.

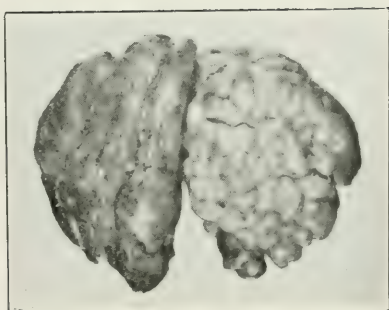


Fig. 1.
Tumor attached to tonsil.

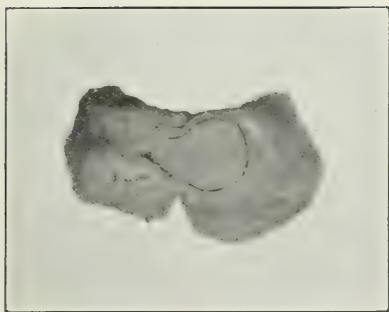


Fig. 2.
Cross section through the pedicle.

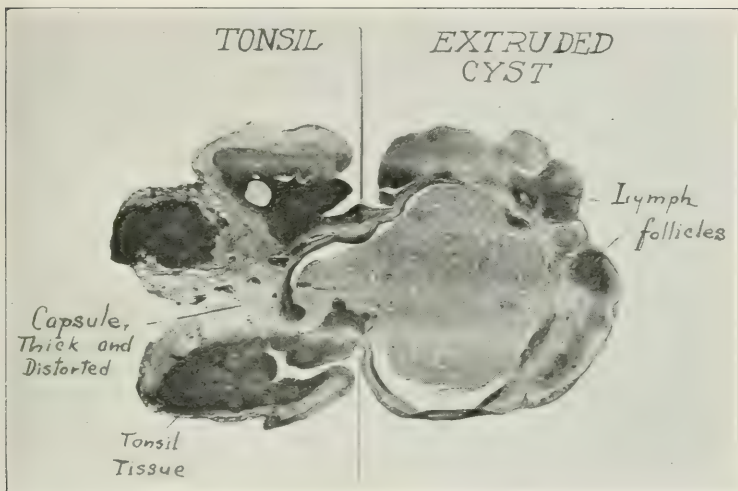


Fig. 3.
Section through the pedicle.

XVI.

REPORT OF A CASE OF CODEIN POISONING.

BY ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

On June 23, 1920, patient Miss L. B., clerk, age 28, presented herself for treatment. She complained of a dry non-productive cough which had persisted for two weeks. It was apparently not preceded by any acute infectious process, there having been no sore throat, no influenza, or any other systemic disease which might have accounted for the symptoms. Nasal examination was negative, submucous resection having been performed the year before. The pharynx was mildly congested, larynx and trachea were not involved. The diagnosis lay between habit cough and irritation by a slightly elongated uvula. While observing the case the patient was given a prescription for elixir terpine hydrate with codein (N. F.), in which the codein content is one grain to the ounce. Dose: One teaspoonful every three hours.

After taking three consecutive doses (about one-half grain of codein) the patient complained of lassitude, some nausea, cold and clammy skin, and general apathy. The mixture was discontinued and the following day the patient felt quite well. The cough subsided after a few days under local treatment.

The cause for the patient's indisposition was not determined at the time, and the codein was not suspected until January, 1922, when the patient appeared on a Sunday afternoon complaining of severe toothache. She was unable to find her dentist at that time and asked for something to relieve her pain. As a temporary measure she was given a prescription for codein sulphate, one grain, to be mixed with milk sugar and divided into three capsules. She took one of these capsules and within half an hour exhibited the same symptoms as before: great languor, pallor, increase in pulse rate to 90, skin cold and clammy. It was only then that the similarity of the symptoms to those of the previous attack was noted and

the cause laid to the codein. The patient has never had similar attacks before nor since. In each case the dose was, if anything, less than average.

Wood in his work on therapeutics outlines practically the symptoms given as the result of codein poisoning. He adds that "In S. Weir Mitchell's experiments upon himself five grains produced no symptoms except slight increase in the pulse rate, nausea, some giddiness and a sense of heaviness about the head; results which are in accord with the earlier experiments of Harley. Contrariwise, A. S. Myrtle records a case of severe poisoning caused by four grains of codein. There was first vascular excitement and exhilaration, then depression with great anxiety, nausea and vomiting, pale, cool, clammy skin, slight contraction of pupil and sleeplessness, with slight delirium. Two cases of serious poisoning by eight grains have been reported."

Toxic effects following small doses of codein, although unusual, should be borne in mind, as they may readily be confounded with symptoms of influenza or the toxemias associated with other infections of the upper respiratory tract. In the above case it was only the patient's freedom from any acute infections which fastened the attention upon the codein as the cause of the trouble.

XVII.

REPORT OF TWO CASES OF SALIVARY CALCULUS.

BY ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

Case No. 1.—Miss J. B., a nurse, 24 years old, applied April 20, 1920, for relief from a large but only moderately painful swelling in the right submaxillary triangle of the neck. This swelling appeared quite suddenly during the night, and by the following noon filled the entire triangle, with considerable bulging. The patient had had no symptoms at any time previously and thought she had the mumps. The orifice of Wharton's duct was more prominent on the affected side, but not red. At short intervals small drops of thick, viscous saliva escaped from it. Palpation disclosed an indefinite mass quite far back in the duct. With a small bistoury the duct was slit for about an inch without encountering obstruction. On probing further with a whalebone filiform the stone was encountered and pressure upon it with the bougie, causing a backward dislodgement, produced a copious flow of thick saliva. The tube was slit still farther and a large calculus found, lying in a fusiform sack or dilatation of the duct, which was slightly longer than the stone and in which the latter slid back and forth fairly freely. It was removed without difficulty, the wound healed rapidly, and at the end of a year no signs of any further trouble had developed. This stone measured 11.9 mm. by 8.5 mm. by 5.4 mm. and presented a longitudinal crease or slit which permitted the saliva to pass. See figure.

Case No. 2.—The patient, Miss K. G., aged 14 years, first seen September 6, 1921, complained of deafness of variable intensity for four years, high pitched tinnitus and frequent sore throat, although two tonsillotomies had been done. History of discharging ears at some previous time. Measles at six, no other illnesses. The patient was an extremely refractory child. She had not been living for some years with her mother, who brought her, so that an accurate history was not

available. The data germane to this report, however, were as follows: There was swelling with extreme pain in the region of the right submaxillary gland. This pain had persisted for three to four weeks, becoming excruciating at times, especially during meals. Wharton's duct was thickened and palpable throughout its length. The papilla on the infected side was red and swollen. On palpating the duct a small quantity of ropy saliva containing pus appeared at the papilla. The duct was slit and a quantity of thick, viscous material was liberated. There was a mixed infection, principally staphylococcus. No calculus could be made out at this examination. The patient was seen daily and, as the pain continued with periodic evacuations of purulent saliva, further search for calculus was made six days later. The whalebone filiform encountered a small stone no larger than a coarse grain of sand, which was removed. This granule was unfortunately lost.

Following this, the pain and infection cleared up promptly and remained so three weeks later, when the patient was lost sight of.

These two cases are reported because in each the intensity of symptoms was out of proportion to the size of the offending calculus.

The calculus in the first case was among the largest on record, and had caused no symptoms up to the night preceding its removal. This was probably due to the sulcus which traversed one side of it in a longitudinal direction and permitted the saliva to pass. The second calculus, a mere grain of sand, caused excruciating pain for weeks preceding its removal, and two teeth were sacrificed through the efforts of a dentist to overcome the trouble. In this case there was infection present. The patient's mouth had been badly neglected. There were several carious teeth and heavy deposits of salivary calculus at the gingival margins. The first patient's mouth was in excellent condition.

In the cases reported in the literature, of which about 300 have been collected, pain was the outstanding symptom. The "colique salivaire" is frequently described as an intense, sharp pain on the floor of the mouth, the tongue and the side of the neck.

Erdman reports a case in which an attack occurred four years before the removal of the calculus, with a pain-free interval and recurrence eleven days before operation. With the observation in mind that a mere grain of sand may cause severe symptoms of obstruction, the possibility of spontaneous delivery presents itself, especially in cases of long intervals between attacks. The presence of salivary calculi should be carefully ruled out before teeth are sacrificed in an endeavor to reduce submaxillary swelling.

XVIII.

TRAUMATIC FACIAL DIPLEGIA, WITH INVOLVEMENT OF THE SIXTH NERVES, A PORTION OF THE LEFT THIRD NERVE, ALSO THE FIFTH AND EIGHTH NERVES, WITH DISLOCATION OF THE ATLAS, ETC.*

BY GEORGE F. KEIDER, A. M., M. D.,

LAFAYETTE, INDIANA.

Traumatic facial diplegia is apparently a very rare condition. The great war produced but very few cases. A survey of the literature of this subject for the last thirty-five years, made with the help of the librarian of the office of the Surgeon General of the United States Army, has brought to light the few cases on record, none of which parallel in all respects the description of the case which it is my privilege to present to you today, a case which is of interest to the ophthalmologist, otolaryngologist, neurologist and general surgeon alike.

Bilateral facial palsy may be due to a variety of other causes. Patrick gathered together forty-eight cases due to multiple neuritis. Ross found five due to diphtheria and five due to influenza. It may be due to double otitis media and mastoiditis. Throckmorton reported a case due to rheumatism, wherein an interval of a week elapsed before the other side of the face became involved. Sir Charles Bell reported a case due to syphilis.

But the cases due to traumatism are indeed very rare.

Ransohoff, in a communication to the 1919 meeting of the American Surgical Society, could find but four other cases in the literature than his own and appended one with Sach's permission hitherto unreported. He failed to note the earliest case reported by our own lamented confrere, Alvin W. Hubbell of Buffalo. In giving a resumé of the cases reported to

*Read before the twenty-seventh annual meeting of the American Academy of Ophthalmology and Otolaryngology, Minneapolis, Minn., September 19 to 23, 1922.

date I quote liberally and literally from the writing of the reporters.

The first case to be reported, as far as we can find, as intimated above, was in 1889 by Hubbell: Henry M., aged 23, a switchman, while coupling cars, August 21, 1888, had his head caught between the projecting ends of the loaded lumber on two cars, between which he stood at the time. He was picked up unconscious and hurried to the hospital. Hemorrhage poured profusely from both ears, nose and mouth. He vomited blood frequently. Pulse was 54. Diagnosis was given as fracture of the base of the skull. On the following morning he was in a semiconscious state. On recovering consciousness it was found that the hearing of both sides was somewhat impaired. Complete facial paralysis of both sides of the face was noted. The patient was unable to close the eyelids or to move the lips. His face was absolutely expressionless. Both right and left abducens were completely paralyzed, resulting in a marked convergent squint of both eyes. The pupil of the right eye was dilated and its accommodation impaired. The right side of the tongue was dry and rough, with a total loss of the sense of taste. Deglutition was difficult. Sensation and motion in other parts of the body were normal. He had intense pain in the forehead. He gradually improved and his sense of taste returned after a month. The sight and hearing returned to normal. The pupil contracted down.

The reporter on July 2, 1889, stated that the facial paralysis had so far improved that he could nearly close his eyelids and in laughter could move some of the muscles of the mouth. The left abducens had nearly recovered its function. The right abducens was still paralyzed.

For the following four cases I am indebted to Ransohoff in the article mentioned above:

Case 1 (Koslovsky).—Private J. J., white, while carrying logs from a boat to a landing fell in the water, a distance of ten feet, and was struck by a log over the left side of the head. When brought to the hospital he was bleeding from both ears and nose and was vomiting. Consciousness was retained but speech was very indistinct. There was a large hematoma on the left side of the scalp. No other external injuries. He had considerable headache. Temperature was normal, likewise the

pulse. The pupils refused to respond normally to light and shade, but later the pupil became normal in the right eye. The left pupil was dilated and did not respond to light. There was constant and marked diplopia. Hearing was much lessened on the right and entirely lost on the left side. The tuning fork could not be heard on this side. There was complete paralysis of both sides of the face.

All the symptoms detailed above appeared simultaneously. After three months the paralysis of the face continued unchanged. The patient remained completely deaf in the left ear, while the hearing in the right ear was somewhat improved. The sense of taste was at no time affected. When the patient was last seen it seemed that the condition detailed at the last examination would be permanent.

Although there was no X-ray confirmation, the diagnosis of basal fracture could not be questioned. The injury to the facial nerves must have been above the geniculate ganglion, from the undisturbed taste function. It is probable that from the involvement of the other cranial nerves the tear was above the entrance of the nerves into the internal auditory meatus of each side.

Case 2 (Oppenheim and Hallez).—P. J., age 28, infantryman, was injured October 27, 1916, by the bursting of a shell in close proximity. There was immediately loss of consciousness, followed by prolonged anorexia. He could furnish no details as to how he was hurt. Consciousness returned sixteen hours later. The primary reports state that there were no external lesions. Double facial paralysis, bleeding from both ears, profuse nose bleed and slight mydriasis on the left side were all apparent. After a day or two he became semi-conscious again, and when he recovered he complained greatly of nausea and vertigo. Epistaxis and bleeding from one ear returned again. He entered the otologic service two weeks after his injury for "concussion of the labyrinth, injury to the tympana, with infection on both sides, and facial diplegia."

The face presented the appearance of a blank mask, without any expression, the creases of the forehead and the face having completely disappeared, and there was slight edema of the face in both cheeks. There was no diplopia and visual acuity was normal. Speech was a little slow, scanning and especially

embarrassed by the pronunciation of the labials. Deglutition was normal.

It is interesting to note that after the first visit of the patient's wife, in consequence of a violent emotion the man presented a complete mutism for four days.

The cheeks were flabby, mastication was considerably embarrassed, because foodstuffs fell between the gums and the cheeks.

The soft palate was lifted normally, and there was no deflection of the uvula. The sensibility of the tongue was conserved, but the sense of taste was abolished in the anterior two-thirds.

The electrical examination, made in January, two months after the injury, showed a degeneration reaction of the seventh pair a little more marked on the right than on the left side. The hearing on the right was diminished and there was suppuration of the middle ear. On the left side the hearing was quite gone and there was suppuration of the middle ear. The facial and auditory nerves seem therefore to have been involved, but no others were in any way injured.

The lumbar puncture made in January gave a clear lymphoid fluid with a little more albumin than normal and a scarcely appreciable increase of the cellular contents.

The lesion doubtless therefore was in the fallopian canal on each side. After several months' sojourn in the service the patient improved considerably, the eyelids could be partly closed and the forehead could be slightly furrowed. As to the course of the paralysis the authors are in doubt. It might have resulted from a fracture through the petrous portions of both temporal bones, or again it might have resulted from an immediate extratubal concussion. It is also possible that the injured man was thrown backward and sustained a fracture of the base through both temporal bones. In favor of this hypothesis are the nose bleed, the repeated bleedings from the ears and the prolonged coma. In support of the possibility of the shock of the explosion alone causing the damage in the petrous portion of the temporal bones without fracture, the authors allude to two men who, without any trace of external injury or even of a fall, presented simply as a result of the concussion, a total unilateral facial palsy.

Case 3 (Chatelin and Patrikos).—The case when presented was of six weeks' duration. There was a double paralysis of the peripheral type. On the left side the paralysis was absolute, while on the right side there was an indication of very slight movement in an effort to close the eye. The aspect of the face was characteristic. The forehead was entirely without wrinkles, the nasolabial folds had disappeared, the lips were half open and the saliva drooled from the mouth. Speech was almost incomprehensible. Mastication was difficult. Electrical examination showed complete degeneration reaction on both sides. The diplegia was the result of an accident. The wounded man was about to unload a heavily loaded wagon, when it was suddenly propelled backward and his head was caught transversely between the wagon and the wall. Released at once, the man bled from the left ear. The diplegia manifested itself at once. For several days the face was very much swollen and he had many ecchymoses in the temporomalar region. Eating was uncomfortable on account of the trismus. This, however, disappeared in a few days. The writer believes that the facial nerves were injured on both sides by a transverse fracture at the base of the skull, even if there were actual symptoms of injured lesions of the organ of audition. It appears to the writer that the absence of the loss of taste and of the radiographic evidence of fracture of the base in this case, it is more likely that underneath the ecchymoses described in the temporomalar region the facial nerves had been injured by compression after their exit from the stylomastoid foramina.

Case 4 (Marchand).—Soldier, aet. 22, was thrown from an automobile truck August 22, 1917. He had no recollection how the accident happened. There was immediate bleeding from the ears, with epistaxis and bilateral facial paralysis. In the temporal region there was a contused wound. The patient was unconscious for forty-eight hours.

Diagnosis: Fracture at the base of the skull.

On the morning following admission there was considerable palpebral and subconjunctival hematoma. There was also hematoma at the nape of the neck. A lumbar puncture was made on the day of the accident, but in the original report no

result of the examination was given. During six days the punctures were repeated.

He was admitted to the neurologic center at Bordeaux, on December 4, 1917, when the facial paralysis continued. The record states that the movements of the face are nil on both sides. The patient cannot laugh, cry or whistle. Speech is difficult on account of the inability to pronounce the letters P, B and M. Swallowing is normal, but to drink the patient must hold his head backward to prevent liquids from running out of his mouth. The sense of pain and touch is normal, both in the tongue and in the face. Salt, acids or sweets are not felt on the anterior two-thirds of the tongue on the right side, but the taste for bitter things is normal on both sides. In March, 1918, there was some voluntary movement of the lower portion of the left side and the left eye could be completely closed. There was still a loss of the gustatory sense of the left side. On the right side the paralysis presented improvement.

Case 5 (Ransohoff's own).—Miss E. K., aet. 22, was injured January 4, 1919, in a street car accident. She bled from both ears and was unconscious when admitted to the Cincinnati General Hospital. She was in coma for one hour. Vomiting followed. Then two general convulsions followed, lasting for five minutes. Patient suffered from severe headaches with pain in the ear. She had nausea for ten days. The pulse and temperature were normal. Complete left side paralysis was present with slight ecchymosis over both mastoids.

On the morning of the third day after admission the right side of the face became completely paralyzed. She was mentally sluggish. The right pupil was larger than the left. The left external rectus muscle was weak. The other eye muscles were normal. The patient was unable to close either eye or to move the wings of the nose or the lips. The face was mask-like. The lips flapped loosely on expiration. Speech was very indistinct.

Dr. Iglauer examined the ears. The right ear showed slight tinnitus. The drums were intact. There was no spontaneous nystagmus. Weber was lateralized to the left. Bone conduction was normal. The whispered voice was heard at eight

feet. No hyperacusis was present. The eighth nerves were intact.

She could protrude her tongue in the midline and move it from side to side. The uvula hung in the midline. The sense of touch was normal. The sense of taste was absent on both sides of the tongue in its anterior two-thirds. She could not distinguish sweets, vinegar or bitter things.

The X-ray showed an oblique fracture through the cranial base running through the left mastoid process into the petrous portion of the temporal bone.

Paralysis disappeared from the right side of the face in two or three weeks, and during the next two months it disappeared entirely from the left side. Muscular control began in the lower portion of the face. The eyelids recovered last. Both sides recovered alike.

The lesion in Ransohoff's case was in each fallopian canal below the geniculate ganglion and at a point above the point where the chorda tympani is given off. It was also below the point where the nerve to the stapedius is given off, since there was no record of hyperacusis.

Ransohoff refers to the theory of Moure, that the facial nerve in the fallopian canal is composed of two sets of fibers: one peripheral or superficial, which supplies the lower facial group of muscles, and the central or deep, which innervates the upper muscular group. But there was never any anatomic demonstration of his theory, and the case of Ransohoff denies such an hypothesis.

Case 6 (Sachs).—Patient, aet. 16, admitted October 11, 1912, after a motorcycle accident. He was bleeding from the right ear and nose. No fracture could be made out by palpation. K. K. was negative. There was no ankle clonus and no Babinski reflex present. The left pupil was somewhat dilated. It did not respond to light. The left eye showed slight exophthalmos. Pulse was 138. Blood pressure was 135 mm. The vessels of the eye grounds were tortuous. Lumbar puncture could not be made. The right external rectus muscle was completely paralyzed and the left one slightly so. He could not pucker his lips or whistle. Neither facial nerve reacted to faradism. The strongest currents possible produced contractions, however. Quinin was not tasted in the anterior

portion of the tongue, but he did recognize it on the posterior portion of the tongue. Salt and sugar were recognized. When asleep both eyes closed, but when awake he could not close them. X-ray plates did not show basal fracture. On discharge October 11, it was noted that for the first time he was beginning to gain the use of the muscles about the mouth on the left side."

This completes the cases referred to in Ransohoff's paper.

Levy's and Tupper's Case.—Colored soldier, aet. 24, hurt June 9, 1919. His head was caught between two railroad cars. He was unconscious for three weeks. On August 23, 1919, he entered Cook County Hospital, Chicago. Examination revealed excellent mental and physical condition. No Romberg was present. There were no abnormalities in gait or in muscular, bone or genitourinary systems. No headache was present, neither dizziness nor gastrointestinal derangement. He could not pronounce labials.

The face was expressionless and the nasolabial folds were obliterated. The corners of the mouth hung down, especially the left. He could not wrinkle the forehead, pucker the lips, close the eyes or show his teeth. To drink he must roll up the lower lip and support it. The tongue was very dry and corrugated, but it could be protruded and moved in every direction. The uvula and soft palate were normal. The left eye showed marked paralysis of external movement. There was no nystagmus.

The sensation for taste, sour, sweet and salt, was lost over the anterior two-thirds of the tongue. The sensibility of the face and tongue was normal. All reflexes were normal save the corneal and conjunctival. Electrical examination showed degeneration of both seventh nerves and of the muscles supplied by them.

X-ray examination (Dr. Blaine) showed a shadow indicating fracture into the middle fossa of the skull close to the base and involving the right parietal temporal region.

Nystagmus tests: Rotation to the right produced no nystagmus. To the left, six seconds of nystagmus were produced, but to the right. There was no past pointing after rotation to the left. But after rotation to the right the patient past pointed two inches to the right with both hands. Hearing was

fairly good. On October 9, 1919, the right ear was douched with cold water, with no reactions from either horizontal or vertical canals. There was no past pointing, vertigo or nystagmus. Douching the left ear produced no response from the vertical canals. The horizontal canals gave slow nystagmus of fifteen seconds and past pointing to the left. On October 13, 1919, the vertical canals were stimulated by rotation and the reactions were normal but shortened. Stimulated by rotation to the right the horizontal canals gave nystagmus to the left, lasting for three seconds, and momentary past pointing with the left but not the right hand. Hearing was normal in the left ear, but somewhat diminished in the right ear. Bone conduction was normal on the left side but prolonged on the right side of the head. Conclusion: "The lesion of the vestibular apparatus is not due to a labyrinth destruction, is not peripheral."

Course: The right facial nerve, especially the lower branch, showed marked and progressive improvement, but the left facial nerve and the left abducens remained stationary at the time of the last examination.

Summary and Discussion.—1. There was bilateral degenerative facial paralysis of the peripheral type. 2. There was paralysis of the left abducens. 3. The chorda tympani was involved. 4. On the right side the hearing was impaired. 5. The lesion was one of the central portion of the vestibular apparatus.

The chorda tympani speaks for the involvement of both facial nerves in their fallopian canals—i. e., in the petrous portions of the temporal bones. But bilateral fractures of the pyramid bone is usually fatal (Marchand) and could not explain the involvement of the sixth nerve. We look for several or multiple lesions which are the rule in severe concussions of the brain. (Jacob.)

The writer's case:

Oscar Gustafson, Swede, aet. 34, was caught in a cave-in while laying sewer pipe in a deep trench. He was completely buried and when dug out was unconscious and bleeding from both ears and nose.

He regained consciousness in a few hours, and it was observed that his face was completely paralyzed. Complete internal squint of the left eye was present and partial of the right eye. He had diplopia. The record shows that he was very restless, complaining of severe pain in the head. The upper portion of the right chest was painful. At eight the night of the same day he was given 1,500 units of anti-tetanic serum.

His past history reveals nothing of the diseases of childhood, save measles. He had influenza two years before. Otherwise he has always been healthy. There is no history of lues and the Wassermann tests are negative. Neither tuberculosis nor insanity nor cancer affected any member of his family, past or present. His father died at the age of forty of pneumonia. His mother is living and well at the age of seventy-seven. One brother is living and well at the age of forty. Two sisters are living and well at the ages of forty-five and fifty, respectively.

Examination of his person: The right clavicle was fractured. Below the right ear was a perforating wound with partial separation of the auricle from the head. Bloody spinal fluid was obtained at a pressure of 150 mm.

The face was expressionless. He could not show his teeth nor whistle. He could not close his eyes. His speech was very indistinct because of the lack of lip action. Internal strabismus was very marked in both eyes. His tongue and lips were quite dry. He had trouble eating and drinking, because the food and drink would drop in between the cheeks and the jaws.

Sensation to touch and pain remained normal.

The thorax expanded fairly well and the breath sounds were of good quality and there were no rubs or rales.

The left border of the heart was well within the nipple line. No murmurs, shocks or thrills were present.

The abdomen was slightly tender above the symphysis and there was no distension and no tumor masses could be made out.

The genitourinary apparatus was normal.

The skin was clear.

The joints were flexible.

The costochondral end of the right fourth rib was depressed and pressure caused pain.

There was no adenopathy.

The biceps, knee jerk and other reflexes were normal.

The pupils were equal and responded fairly well to light and shade.

X-ray examination revealed a dislocation of the atlas on the axis. Through the pedicle of the fourth cervical vertebra was an oblique fracture and a fracture of its spinous process.

The soft palate and uvula were normal. He could protrude his tongue and move it in every direction.

While the X-ray plate was not satisfactory, yet it seemed to show a fracture of the base of the skull passing through both temporal bones.

His temperature and pulse were as follows: July 5th—Temperature, 100.4°; pulse, 64. July 17th—Temperature, 97.6°; pulse, 50. July 19th—Temperature, 98°; pulse, 48. Otherwise both ran a normal course.

Both ears discharged pus freely.

On July 21st, on account of the eye condition, I was asked to take charge of the case by Dr. A. C. Arnett, his physician.

All the above conditions were present on that date, and in addition it was observed that the upper lid of the left eye drooped markedly. Both eyes have marked limitation of motion temporally.

Hearing tests were made. Weber was not lateralized. Rinne is negative. Right ear: Bone conduction, + 20 seconds, and air conduction, 14 seconds. Left ear: Bone conduction, + 25 seconds, and air conduction, 22 seconds. The tuning fork has 128 double vibrations per second and is weighted to guard against overtones. A stop watch was used as a timer.

Both membranæ were ruptured and pus was present in the lips of the wounds.

The pupils were normal and responded well to light and shade. The fundi appeared normal.

Protecting glasses with wire mesh temple guards were ordered to protect the eyes against extraneous substances of which the paralyzed eyelids could not dispose.

On July 21, 1921, tests of sensation were made with the esthesiometer with the following results:

	Right	Left
Supraorbital	6 mm.	9 mm.
Infraorbital	6 mm.	16 mm.
Upper lip.....	4 mm.	5½ mm.
Mental	7 mm.	10 mm.

The sense of smell seemed unimpaired.

The sense of taste was markedly impaired. He did not and does not recognize either quinin or vinegar placed anywhere on the tongue. Sugar and salt were barely recognized on the posterior portion of the tongue but not on its anterior portion.

On August 3rd he was allowed out of bed.

September 15th we made a record of his hearing as follows: Weber not lateralized. Rinne—Right ear, bone conduction, 25 seconds; air conduction, 32 seconds. Left ear, bone conduction, 32 seconds; air conduction, 28 seconds. Range of hearing, 128 d. v. s. to 38,000 d. v. s.

For the sake of comparison I will here insert the record of the last tests, made June 26, 1922:

Weber is now lateralized to the right. Rinne—Right ear, bone conduction, 22 seconds; air conduction, 51½ seconds. Left ear, bone conduction, 23 seconds; air conduction, 20 seconds. Low tones, 128 d. v. s. The upper limit is 40,000 d. v. s. Both ear drums are healed over.

Turning tests resulted thus: To the right ten times, nystagmus 20 seconds and of good amplitude. To the left ten times, nystagmus 19 seconds and of good amplitude. Falling tests are normal. He past points normally with both arms. No douching tests were made because of the condition of the ear drums. There were no fistula symptoms.

On September 15, 1921, the tests for sensation were repeated with the following results:

	Right	Left
Supraorbital foramen	8 mm.	10½ mm.
Infraorbital foramen.....	6½ mm.	9½ mm.
Mental foramen.....	5 mm.	14 mm.
Inframaxillary	15 mm.	18 mm.
Preauricular	11 mm.	12 mm.

The last tests were made August 3, 1922, as follows:

	Right	Left
Supraorbital foramen	9 mm.	10½ mm.
Infraorbital foramen.....	9 mm.	5½ mm.
Mental foramen.....	9 mm.	8 mm.
Tip of tongue	3 mm.	

The left eye gave us a great deal of trouble. Imperfect closure of the eyelids produced exposure keratitis. Iritis began to show itself August 18, 1921, with ulceration of the cornea, just below the center. Atropin and bandage with hot applications controlled the pathologic condition pretty well. Lugol's solution of iodine was also applied locally. By September 11th the healing was complete, with the usual scar. The ulcer recurred October 14th, and although the same treatment was pursued yet it progressed, and on October 26th we were compelled to raise a Kuhnt flap to prevent perforation of the cornea. The stitches pulled out on the 28th, but the ulcer had practically healed over. October 29th the cornea threatened perforation: Descemet's membrane only was intact. So another Kuhnt's flap was raised and the ulcer covered. On November 2nd, the stitches pulled out, but by the 7th the healing was practically complete.

Prince's method of pasteurization was tried, without avail, as well as thermophor of Shahan, likewise without results.

Inasmuch as others had made zinc fluorescein and mercury fluorescein (mercurochrome), not to be outdone I proceeded to make silver fluorescein by mixing a 2 per cent solution of silver nitrate with an equal quantity of the usual fluorescein used routinely in our offices to outline corneal ulcers. This was used to combat the intercurrent suppuration, but it availed naught.

On December 23rd the ulcer recurred with rapid thinning of the corneal substance. We raised another Kuhnt flap from below again and covered the ulcer. It was effective in preventing perforation. But the ulcer did not heal in well this time, and again on January 4, 1922, we raised another Kuhnt flap because the ulcer threatened perforation. This was the last one needed, for the eye cleared promptly and the inflammation left it completely. Of course there is a large scar extending

up into the pupillary area, about one-half being occupied thus. Blood vessels can be seen coursing it from the limbus upward, nature's way of healing the ulcer.

Recession of the eyeballs into the orbit was noted in due time, and on November 29, 1921, I made measurements with the exophthalmometer of Hertel. The right eye measured 12 mm. and the left eye 11 mm. I checked this result with two normal individuals. One was the male attendant on the floor of St. Elizabeth Hospital, where our patient was confined, and the instrument measured 20 mm. for the right eye and 18 mm. for the left eye. Sister A.'s eyes showed 18 mm. for each eye. At the present the exophthalmometer measures 11 mm. for each eyeball.

The vision for the right eye, June 26, 1922, is 20/20, which is increased to 20/15 with a plus .50 cylinder axis 90 degrees. The vision of the left eye is 20/50 and no lens helps.

The blood examination was and is normal and so is the urine test.

The bacteria present in the conjunctival sac were the staphylococcus pyogenes albus and aureus and the micrococcus catarrhalis.

He is gradually improving and at the present his face is more mobile. He can now close his eyes pretty well. His speech is more distinct, as he has gained some control over his lips.

This patient was treated by an osteopath while under my care. That there may be no vainglorying, I had X-ray plates made September 1st, 1922. The dislocation of the atlas upon the axis still persists. Furthermore, a distinct line of separation of the parietal and occipital bones is apparent. The line of fracture into the petrous portion of the temporal bone still shows.

Observations in general as to this patient:

1. What is the meaning of the hemorrhage from the nose and ears?
2. What is the meaning of the ptosis of the left upper eyelid?
3. What is the meaning of the loss of hearing and its recovery in part?
4. What is the meaning of the double facial paralysis?

5. What is the meaning of the abducens paralysis?
6. What is the meaning of the reduced unilateral sensation?
7. What is the meaning of the apparent recession of the eyeballs into the orbit?

1. As he has survived, detailed examination of the cranium and its contents is impossible. But he suffered a fracture of the base of the skull involving, no doubt, the petrous portions of the temporal bones and probably the lesser wing of the sphenoid. The skull must have endured intense lateral pressure.

2. The ptosis of the left upper eyelid is hard to explain, because the section of the third nerve supplying the eyelid lies so far in advance of the other divisions of the nerve, as the lantern slide will show. Yet the median longitudinal fasciculus connects all the nerves involved.

3. As the static portion of the labyrinth was not involved, according to the tests applied, the cochlear portion was damaged somewhere between the internal auditory meatus and the origin of the nerves. However, this is pure speculation. The vestibular tests indicate that there was no involvement of the pons.

4. The double facial paralysis may be explained in a number of ways. It is probable that the basal fracture may have compressed both nerves in their fallopian canals. Or the corresponding hemorrhage compressing one nerve may have passed over from one nerve to the other, as the lantern slide will show. Again, the hemorrhage may have been in the nuclei of origin.

5. The involvement of the abducens nerves is readily demonstrated by the lantern slide. It will be observed that the nucleus of the sixth nerve lies under the origin of the seventh nerve. Moreover, the fibers of the seventh nerve wind around the nucleus of the sixth nerve in the floor of the fourth ventricle. This tends to prove that the origin of all his trouble thus is a hemorrhage into the nuclei of origin.

6. The reduced unilateral sensation all over the left side of the face may be explained by a consideration of the anatomy of the fifth nerve, and I quote freely from Deaver's Surgical Anatomy of the Head and Neck. "The fifth cranial nerve, trigeminus or trifacial, the largest of the cranial nerves, arises

from the sides of the pons by two roots, a large posterior or sensory root, and a smaller anterior or motor root. These can be traced to the floor of the fourth ventricle and to the gray matter in the lower part of the medulla oblongata and in the upper part of the spinal cord. It is the only cranial nerve which resembles a spinal nerve in arising from two roots. The trifacial is a mixed nerve. It distributes sensory filaments to the dura mater, pia mater, orbit, eyelids, nose, gums, teeth, tonsils, palate, sphenoid cells, ethmoid cells, frontal sinus, maxillary sinus, nasal fossæ, pharynx, articulation of the lower jaw, ear, parotid gland, scalp, forehead, face and gustatory filaments to the anterior two-thirds of the tongue." I may state, however, that the distribution to the tongue is otherwise described by other anatomists. The same hemorrhage into the nuclei of origin of the other nerves involved also spread to involve the origin of the left fifth nerve, damaging also his sense of taste.

7. The recession of the eyeballs into the orbit may be explained as due to a trophic change going on in the orbit, probably absorbing some of the fat therein.

Ultimate Conclusion.—Because postmortem examination is impossible, we cannot determine the exact lesion and its location. But when all observations are put together we are almost forced to the conclusion that the nuclei themselves in the floor of the fourth ventricle must have been affected by hemorrhage which as it is absorbing is bringing recovery to the parts affected.

According to Cunningham, the anterior two-thirds of the tongue is supplied by the chorda tympani so far as the taste buds are concerned. The posterior two-thirds is supplied by the glossopharyngeal. The epiglottidean taste buds are supplied by the inferior laryngeal nerve. For general sensation the tongue relies upon the lingual branch of the mandibular division of the fifth nerve.

A remarkable feature of all these cases reported to date is that not one had perished. Hence we are driven to the conclusion that the prognosis is reasonably good, for improvement at least.

Our Compensation Laws.—This patient revealed to me some phases of our compensation laws not noted heretofore. As a

rule, the laws of the various states provide a maximum of sixty days' hospital care and physician's services. Patients injured thus cannot recover in a period of sixty days. Hence the insurance company can hide behind this phase of the law and refuse to pay either the hospital or doctor for more than sixty days' services. Such patients are laboring on comparatively small pay. As matters now stand, the doctor is compelled to stand for what the insurance company should pay. Let us go to our homes in the various states to see that the compensation laws are amended that these companies assume the entire cost of medical and hospital care for these unfortunates, for their pay goes on in a measure until recovery is acquired. We should receive our just dues.

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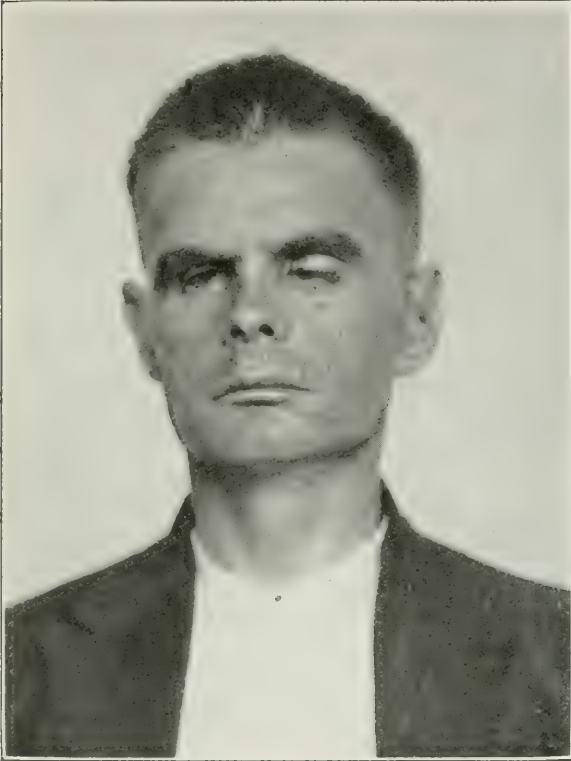


Fig. 1.

Oscar G. His face in repose. Utterly devoid of expression.

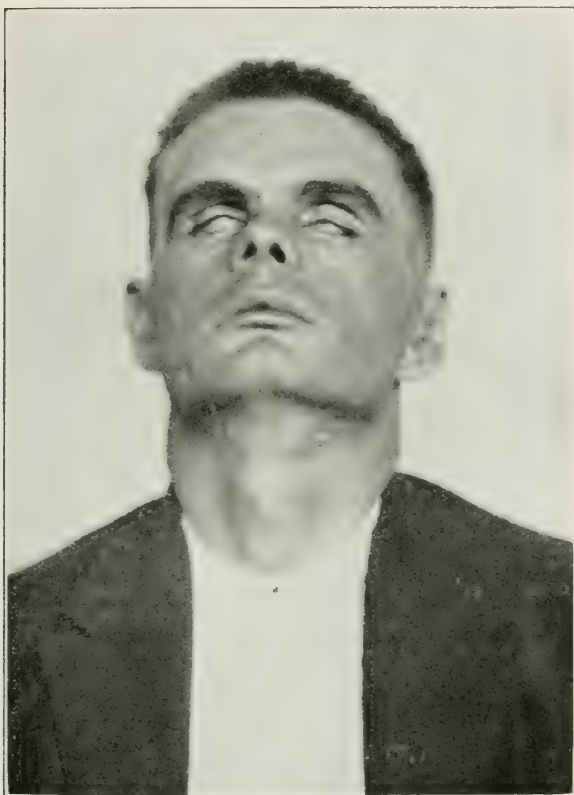


Fig. 2.
Oscar G. He cannot close his eyelids.

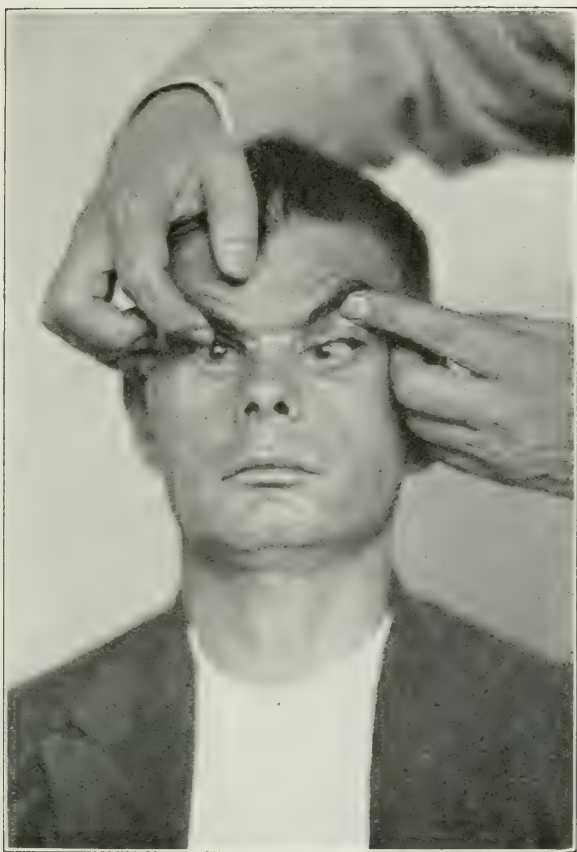


Fig. 3.

Oscar G. The left eyeball inturns markedly and the right one partially.

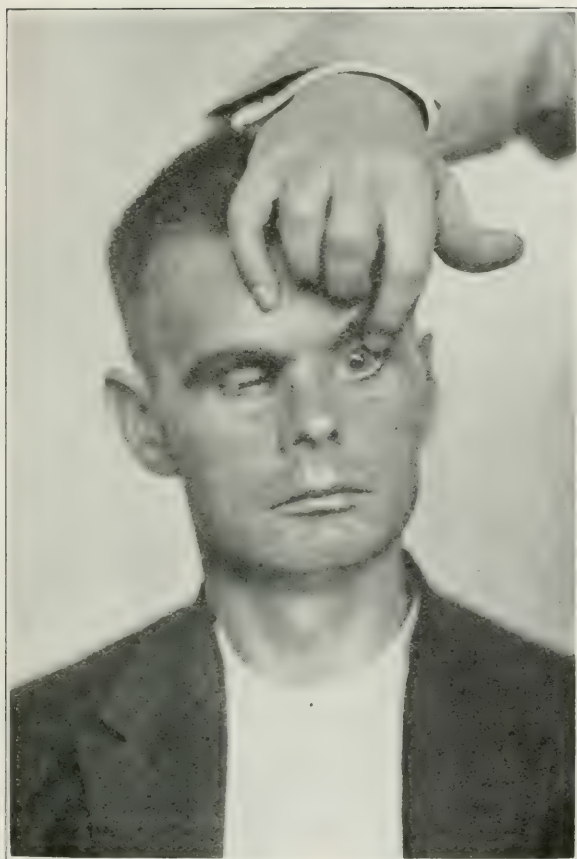


Fig. 4.

Oscar G. The left eyeball refuses outward rotation.

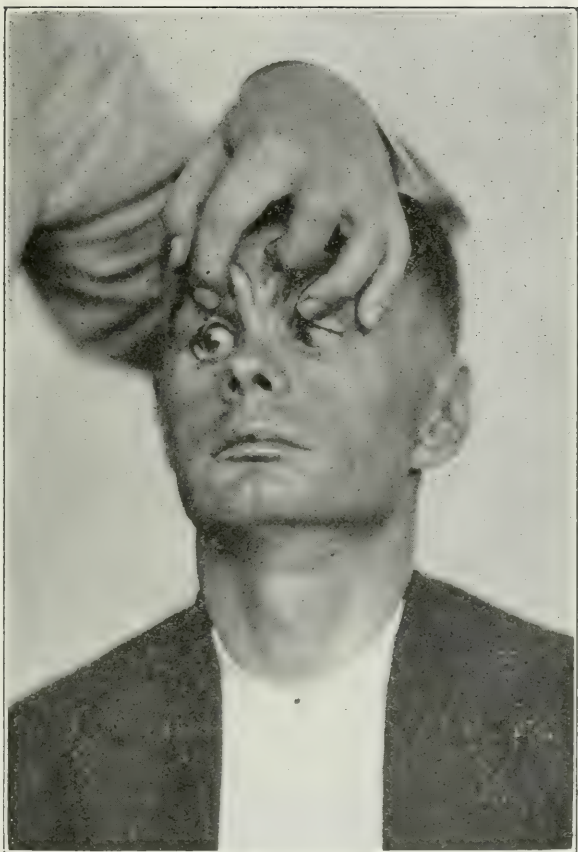


Fig. 5.

Oscar G. The right eyeball refuses perfect outward rotation.



Fig. 6.

Oscar G. X-Ray plate taken shortly after the accident, showing the dislocation of the atlas upon the axis.



Fig. 7.

Oscar G. X-Ray plate taken Sept. 5th, 1922. The dislocation of the atlas upon the axis still persists.

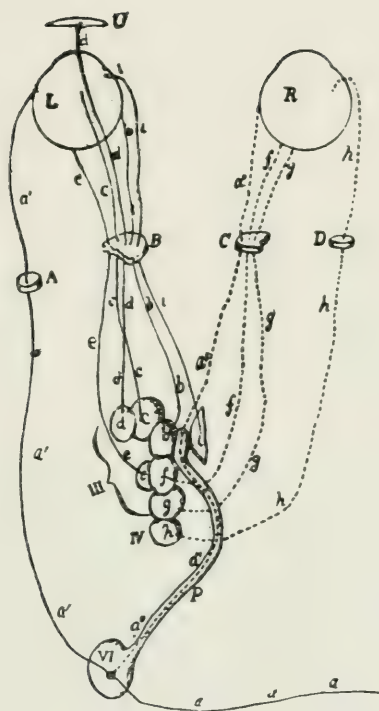


Fig. 8.

Oscar G. To show the distribution of the filaments of the third nerve.

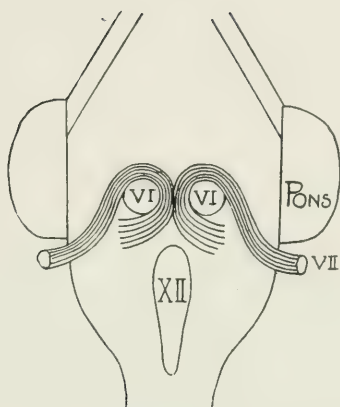


Fig. 9.

Oscar G. Shows how the sixth nerves may be involved with the seventh nerves, at point of origin.

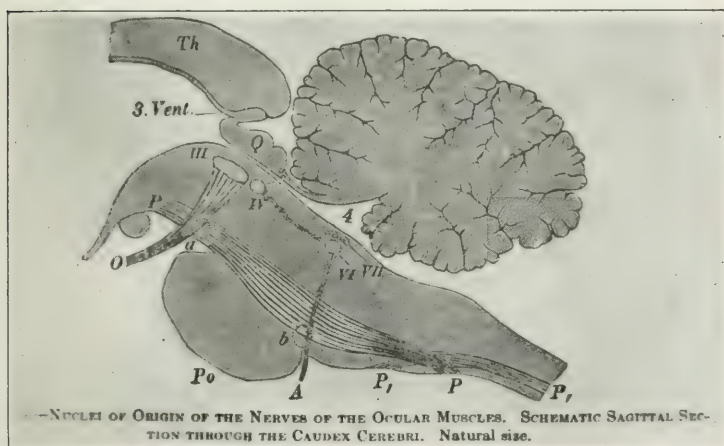


Fig. 10.

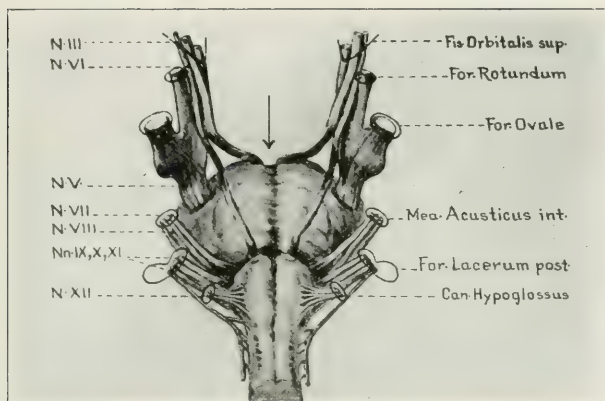


Fig. 11.

Oscar G. How easy for a hemorrhage to pass from one seventh nerve to the other. From Cushing's "Tumors of the Acusticus."

XIX.

TWO CASES OF EMPYEMA OF THE MAXILLARY SINUS OF DENTAL ORIGIN. ALVEOLAR FISTULA. RADICAL AND PLASTIC OPERATION.

BY J. L. MAYBAUM, M. D., F. A. C. S.,

NEW YORK.

The difficulty of successfully eradicating a fistula between the maxillary antrum and the mouth suggested itself as a sufficient reason for presenting these two cases. With few exceptions, these cases are due to alveolar necrosis with antrum involvement or, at least, they are cases of permanent sinus following an operation for antrum suppuration secondary to alveolar necrosis.

Rarely a large infected dentigerous cyst lying external to the antrum and secondarily infecting it may be responsible for the condition.

An alveolar sinus of small caliber responds readily to treatment by the use of cauterizing agents, provided, however, the antrum infection had been eliminated. The problem of obliterating a sinus of larger size is a more difficult one. This can only be accomplished by means of some form of plastic operation, together with a radical antrum operation to provide for thorough drainage through the nose. While little or no difficulty is found in clearing up the antrum suppuration, because of the fact that sutures as ordinarily used are likely to loosen or tear through the mucous membrane, a number of operations may be necessary before the fistula is finally healed. In order to obtain a successful result at the primary operation, it is necessary in the first place to remove enough of the alveolus, having previously separated the mucoperiosteum sufficiently to allow of easy approximation of the flaps without tension, and secondly the sutures are to be applied in a manner to be described, so as to avoid tearing through the flaps.

Case 1.—E. S., age 26 years, referred to me by her dentist, with the following history: Seven months previously she

had the left second bicuspid and first molar teeth extracted. From then until the time I first saw her she had been complaining of more or less constant pain in the left antrum, headache, foul smelling discharge from the nose and a bad taste in her mouth. Patient insisted that she had lost about twenty-five pounds during this period.

Examination of patient: Poorly nourished, anemic individual. Alveolar sinus (left) circular in outline, half inch in diameter. Thick purulent discharge flowing from sinus into mouth. Probe entered the antrum for a considerable distance to the orbital wall; rough bone in sinus wall. Examination of nose disclosed purulent discharge in middle meatus, and on irrigating the antrum through the alveolar sinus pus flowed freely from the left nasal chamber. The only treatment the patient had received from her dentist throughout the seven months had been saline irrigations and packing the fistula on alternate days. The technic I employed as regards the antrum operation differed in no way from the classical Caldwell-Luc operation, except that the gingival incision was made at a higher level than usual, in order to allow for a fairly good size flap in attempting to close the large fistula.

The procedure of closing the fistula consisted of extending the incision through the mucoperiosteum from the margin of the fistula in an anterior and posterior direction for a distance of one inch. I then elevated the mucoperiosteum on the inner and outer surface of the alveolus, removing enough of the latter so as to readily bring together the two flaps. Black silk, No. 4, was used as suturing material. Suppuration from the antrum ceased entirely after a time, but sutures closing the fistula became loose, so that the fistula was as widely open as before the operation. In consulting the literature on the subject, I found that Dr. L. W. Dean of Iowa City had after many trials succeeded in devising a method of procedure which resulted in successful closure of these sinuses in a majority of his twenty-three cases at the primary operation.

This method I followed at the second attempt to close the fistula, with complete success. Briefly, the method is as follows: If the operation is done at the time of the antrum operation, as it ordinarily should be, the incision for the

Caldwell-Luc is made high up and entirely separate from the incision on the border of the alveolar process. After completing the Caldwell-Luc operation and being sure that there is excellent drainage into the nose, the alveolar process is attacked. The necrotic bone is completely removed, and enough of the inner and outer lamella of the alveolar process is removed to allow of perfect apposition of the flaps that have been previously lifted from its inner and outer surface. It is necessary to have these flaps come together nicely in order to avoid any tension on the sutures. Silkworm sutures are used instead of silk, as these can be kept clean and hold several days longer than silk. At this point a piece of small rubber tube sufficiently long to allow it to be tucked underneath all the sutures is placed on the inner and outer surface of the alveolar process.

Using double armed silkworm sutures, a sufficient number are introduced from the inner flap and through the outer, the loops resting on the rubber tubing on the inner surface of the alveolar process, the knots being tied on the rubber tubing on the outer surface of the alveolar process. The sutures should be tied tight enough, without getting them so tight as to produce pressure necrosis of the flap. The alveolar sinus in the case of my patient was completely healed one week after the operation.

Case 2.—The problem in this case was far more difficult than that of the previous one.

The patient, age 38 years, gave a history which differed in no respect from that of the previous case, except that six weeks following the extraction of three teeth her dentist had found it necessary to remove a considerable amount of bone from the alveolar process and the anterior wall of the antrum, because of an extensive necrotic process. For a period of four months following this the only treatment she had received was daily irrigations and packing.

On retracting the upper lip, I found a large defect, circular in outline, involving practically the entire anterior wall of the antrum. The interior of the antrum, containing foul smelling pus and granulation, came readily into view. Because of the extensive loss of tissue I offered little hope of completely closing the opening.

Under local anesthesia the interior of the antrum was carefully curetted and a large opening made below the inferior turbinate. I then elevated the mucoperiosteum for a distance of three-quarters to one inch from the border of the large defect and made an incision, as in the previous case, in a posterior and anterior direction. Silkworm sutures were used to bring the flaps together; this could barely be accomplished. Some of the sutures loosened after a few days; the opening, however, was reduced about one-third its former size. A second attempt at closing the opening was made six weeks later, with a further reduction in size. At the present time examination discloses an antrum no longer suppurating and considerably smaller in size, and an opening above the alveolaris one-quarter of an inch in diameter. This opening can be closed without any further trouble. Had the method followed in the first case been known to me and used in this case the large opening in the antrum would have been completely obliterated in all probability, at the first operation.

1 WEST 70TH STREET.

XX.

CHRONIC SUPPURATIVE OTITIS MEDIA.

BY HERRMANN E. BOZER, M. D.,

SECTION ON OTOLARYNGOLOGY AND RHINOLOGY, MAYO CLINIC,

ROCHESTER, MINNESOTA.

During the treatment of chronic suppurative otitis media the question often arise, "How long will the ear remain in good condition after it has become dry and the pathologic condition has been arrested, or apparently cured, and what are the factors that will tend to cause it to revert to its previously diseased condition? This is important, both to the patient and to the physician. The patient quite often is under considerable expense and, after having obtained an apparent cure, is anxious to know how permanent it will be. The physician is called on to give an opinion and it is his duty to advise the patient, not only concerning the future care of the ear and the prevention of recurrences, but to render a fair prognosis as to the permanency of cure.

I have studied the results in 190 selected cases in which treatment was given for chronic suppurative otitis media in the Mayo clinic. In none of these cases was chronic sup-puration influenced by malignancy, mycosis, or any constitutional disease, such as diabetes, active tuberculosis, or syphilis. All of the patients had been treated within a period of one and one-half years, the shortest time since treatment being one year. All of these patients had continued treatment until it was felt that no further benefit could be derived therefrom. In the majority of cases, the disease process had apparently been controlled so that the affected ear had remained dry for at least one week before the patient was dismissed from the clinic. The smaller group of patients had not responded so well, there being still a little moisture in the middle ear at the time of dismissal. Patients on whom the physician was permitted to use every available means have not been included in this study.

The treatment was essentially a combination of the anti-septic, alcohol and caustic methods described by Politzer, varying in each case with the type of the disease process. All obstructions to drainage, such as crusted adherent bands and necrosed ossicles, were removed as far as possible and daily cleansing of the middle ear and attic regions was instituted. This was accomplished by filling the ear with hydrogen peroxid and allowing it to remain for several minutes. The ear was then carefully wiped dry with cotton on an applicator. A 50 per cent solution of alcohol, containing 3 per cent of boric acid and .05 per cent mercuric bichlorid was then instilled into the ear and allowed to remain a few minutes. This also was carefully wiped out and the drying process completed by directing air under pressure into the aural cavity. Care was taken that the current of air was not strong enough to produce vertigo by causing too rapid evaporation. No other type of treatment, such as vaccin therapy, ionization methods, roentgen ray or radium, was used. Any disease in the nose and throat was carefully eliminated as far as possible, and the patient's general condition thoroughly investigated and cared for. The extent of suppuration in the affected ears of all the patients was carefully graded clinically at the time of treatment. The method of classification is purely empirical and is based on the extent and location of the disease process in the ear. In the main, four types of suppuration were recognized.

Type 1 includes cases in which the discharge was thin, viscid, and mucoid, and arose solely from the eustachian tube. The perforation generally is quite large and is usually situated in the anterior inferior quadrant, although at times the whole of the tympanic membrane may be absent, owing to a former active suppurative process of the middle ear, which has since healed.

Type 2 includes cases in which the suppurative process was confined to the hypotympanum and mesotympanum. The perforation of the tympanic membrane may be large or small and situated any place in the pars tensa. The mucous membranes lining the tympanic cavity are often markedly thickened, granular in appearance, and secrete a thick mucoid fluid containing many leukocytes. Areas of necrosis of the bony

walls may be present, and occasionally necrosis of the lower portion of the ossicle may be demonstrated.

Type 3 includes cases in which the suppurative process and necrosis involve the attic as well as the lower portions of the tympanum. Quite often the perforation of the tympanic membrane is in Shrapnell's membrane and most of the suppurative process is concealed from inspection. Generally, however, the attic seems to be involved secondarily, so that Shrapnell's membrane is intact. A probe introduced from below the level of the anterior and posterior folds will find free access into the attic and will quite often dislodge masses of thickened discharge, fragments of necrosed ossicles, and portions of cholesteatoma, which have collected in that region.

Type 4 includes cases in which there is definite suppuration of the antrum and the adjacent mastoid cells as well as suppuration and necrosis of the lower portions of the tympanum. This is quite often demonstrated by fistulous tracts from the posterior canal wall leading into the mastoid cells, the walls of which are often necrotic or have been eroded by cholesteatoma. Occasionally, the aditus ad antrum is so enlarged by cholesteatoma that the anatomic effect of a radical mastoid operation is produced. A radical mastoid operation is indicated in all cases of this type. Preparatory treatment is given before operation with the intention of cleansing the middle ear and its appendages as much as possible in order that the postoperative care may be shortened. An open tube, contributory to the discharge, may be present in any of the foregoing types.

Ninety-six replies were received from 190 patients to whom questionnaires were mailed. In seventy-eight patients (81 per cent) the discharge had entirely ceased and no apparent suppuration was present when the patient left the clinic. In forty-two (54 per cent) the ears remained dry, and in thirty-six (46 per cent) they did not. In eighteen (19 per cent) the discharge had not entirely disappeared at the time of dismissal. In two patients the ears became dry without further treatment, and in sixteen the ears continued to discharge.

Of the forty-four whose ears remained in good condition there were twenty-four males and twenty females. The average

age was thirty-four years. The average duration of the disease before treatment at the clinic was about fourteen years, the time varying from two months to fifty years. Of these patients, twenty had had a constant discharge, while twenty-four had had intermittent discharge, that is, there were intervals during which the ears had apparently been dry. As a rule these periods of quiescence were of short duration, generally lasting only a few months.

Of the patients whose ears did not remain in good condition there were twenty-eight males and twenty-three females. The average age was twenty-four. The average duration of disease was about twelve years, the shortest being three months and the longest thirty-six years. Twenty-one patients had had constant discharge and twenty-nine intermittent discharge.

The sex and age of the patient apparently were not factors in the ultimate results. Constancy of discharge does not seem to be an unfavorable factor. It would also seem that a few years' duration, more or less, would make but little difference in a group in which the duration of the disease is of such length, the average being thirteen years.

The average duration of treatment in the patients who remained in good condition was 3.6 weeks. In those who failed to remain in good condition, the average duration of treatment was 4.7 weeks. Failure to obtain good results cannot be attributed to less effort spent in treatment.

An attempt was made to determine what effect, if any, the etiologic factors in the suppurative process had on the results of treatment. Many cases were of such long standing, however, that the patients were unable to recall the exciting cause. The statistics obtained were thus too limited to justify an attempt to draw any conclusions. It does seem, however, that those cases originating from scarlet fever are very resistant to treatment, and the percentage of failures is as high in this type as in any other type.

As has already been mentioned, an attempt to classify the extent of the disease process of the affected ears was made at the time of the first examination. In order to determine the value of the classification as an aid to prognosis in the results

of local treatment, the ultimate results of treatment in each type are shown in Table 1.

The percentage of good results is no higher in Types 1 and 2 than in the more dangerous, Types 3 and 4. The only factor to cause recurrence of discharge, present in all types to about the same degree, was an open tube. This is the explanation of what, to the patient, is quite paradoxical; that an innocent aural discharge from what was considered an innocent type of ear should recur after apparent cure as easily as that in an ear which is considered quite dangerous. As all these patients could not be examined by us, it was necessary to accept the patient's statement as to the condition of the ear. No doubt many of the Type 3 ears were discharging only because of an open tube, and the attic was still in a good condition. For these reasons I believe that any classification which fails to consider the patency of the tube offers no aid in the prognosis of the permanency of the cessation of discharges; but I am quite sure that such a classification as I have mentioned offers a satisfactory method of measuring the danger to life of any particular suppurating ear.

Lillie¹ has called attention to the fact that those ears in which the functioning membrane has not been completely destroyed are apt to show moisture at times after all active suppuration has been eradicated. Any congestion of the membrane, such as may occur during acute infections of the upper respiratory tract, will cause hypersecretion and result in a moist middle ear cavity. If this membrane has been completely destroyed by the previous suppurative process, however, and the middle ear is lined only by scar tissue, the ear is much more apt to remain dry. This is an element which must also be considered with an open tube in dealing with the prognosis of permanency of cure after treatment of chronic suppurative otitis media.

Occasionally, because of extensive involvement of the attic, it is inferred that the antrum and probably the adjacent mastoid cells must also be affected. Such ears may clear up under treatment and I believe that the two Type 4 ears in this series that responded to treatment were probably of this kind, and that the actual disease process was not so extensive as it appeared to be at first examination.

Lillie² has recently emphasized the importance of exercising care in blowing the nose when there is chronic discharge with patent tube. In this series I attempted to determine to how great an extent this was noticeable to the patients themselves. Table 2 indicates the results obtained in eighty-one patients who were careful not to produce undue back pressure in the nasopharynx and eustachian tube when blowing the nose.

Of eighty-one patients who were careful in blowing their noses 31 per cent reported that this had a decidedly beneficial effect in keeping their ears from discharging. Twenty-eight per cent were sure it had no effect, and 41 per cent were undecided.

There is very little difference in response between the Type 2 ears, which also included the Type 1 and the Type 3 ears. It must be taken into consideration that many of the Type 3 ears also had open tubes and consequently responded to this precaution. This is good reason for suspecting that many of the Type 3 ears, reported to have begun discharging again after apparent cure, were only doing so because of open tubes, and not because the former suppuration in the attics had recurred. These statistics again point to the view that the prognosis of cure from the patient's standpoint, that is, permanent cessation of discharge, cannot be based alone on the actual extent of the suppurative process in the middle ear and its appendages. To a large extent, prognosis must depend on the condition of the tube. This is strictly in conformance with the view of Yankauer.

Ears which have once discharged and have responded to local treatment often become affected during "colds" in the head. Of the fifty-one cases in which the ears were discharging at the end of one year after treatment in twenty-nine the recurrence began with colds. As this included fourteen in which cure had not been complete at the time of dismissal, this number must be deducted. Thus twenty-nine of thirty-seven recurrences (78 per cent) were due to colds, and probably to the consequent nose blowing.

SUMMARY.

About 50 per cent of the patients with chronic suppurative otitis media, who have responded satisfactorily to local treat-

ment through the external auditory meatus, remained in good condition for a period of from one to two and one-half years. An ear that remains in good condition for this length of time holds but little danger to the patient's life, if proper precautions are observed. Many of the patients in this series responded to treatment, although the disease had been of many years' duration. Failure to secure closure of the eustachian tube is a big factor in the failure to keep an ear in good condition after an apparent cure has once been obtained. Care in the manner of blowing the nose is very beneficial in keeping an ear in good condition in about 30 per cent of cases.

CONCLUSIONS.

1. About 50 per cent of the patients with long standing, chronic, suppurative otitis media who have responded satisfactorily to treatment remain in good condition for a period of at least one to two and one-half years.
2. Sex, age, and constancy of discharge have no apparent effect on the permanency of the cure.
3. Long duration of discharge is not necessarily an unfavorable factor.
4. The patency of the eustachian tube and the presence of functioning membrane in the middle ear must be taken into consideration with the type of disease when offering a prognosis as to the permanency of cure.
5. Local treatment must be well directed and persistent.
6. Care in the nose blowing habit is an important factor in keeping an ear dry in at least 30 per cent of the cases; this depends on the condition of the tube and not on the disease in the ear.
7. Many patients whose ears were cured and remained dry would have had a mastoid operation, if a conservative view had not been taken and persistent local treatment kept up.
8. It is reasonably safe to assume that the suppurative ear conditions which have responded well to treatment will do so again.
9. Discharge from the tube, after the suppurative process has been controlled, may be considered of minor importance as far as real danger is concerned.

TABLE I.

Type	Results of Treatment.	
	Good	Unsatisfactory
1	6	6
2	24	27
3	13	15
4	2	3

TABLE II.

Results	Effect on Discharge of Care in Nose Blowing.				
	Number	Per cent	Type 2	Type 3	Type 4
Questionable	33	41			
Good	35	31	14	10	1
No effect	23	28	10	9	4

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XXI.

COMPLICATIONS OF PARANASAL SINUS DISEASE IN INFANTS AND YOUNG CHILDREN.

BY L. W. DEAN, M. D.,

IOWA CITY.

The most serious result of paranasal sinus disease in infants and young children is death.

An infant, two months of age, came into our service in an extremely critical condition, with a hemolytic streptococcic infection of paranasal sinuses and ears. The mastoids were drained with improvement. The paranasal sinus disease was treated. When the infant died the opinion was given that the cause of death was paranasal sinus disease. I was anxious to get the very best possible opinion about this case, so the autopsy was held by Drs. Medlar and Armstrong, the two pathologists of the University Hospital, together. The mastoid wounds were clean. The superior and inferior meati of each side of the nose were filled with large accumulations of pus, which seemed to come from the paranasal sinuses under pressure. In each maxillary sinus pus under great pressure was found. A complete general examination was made with negative findings. The pathologists expressed the opinion definitely that the cause of death was the paranasal sinus disease.

Another infant, six weeks of age, with a similar history died while it was under treatment for paranasal sinus disease of streptococcic origin. Both of these infants were very sick children when they came into our service. They were so sick that it was necessary to feed them by lavage. When an infant two or three months of age comes into our service with a temperature of $105\frac{1}{2}$, due to paranasal sinus disease, and we get the child early before the gastrointestinal disturbances are marked the prognosis is very good. If when the treatment is begun the child has reached a stage where it will no longer take nourishment of itself the prognosis is very bad. This

second infant who died, like the first, had pus under pressure in every paranasal sinus. The pathologist stated that this was the cause of death.

There may be an interdependence between chronic multiple infectious arthritis and paranasal sinus disease in infants and young children. This may be present at the age of three.

On December 4, 1918, Baby E. T., age three, was referred to us by Dr. Steindler for examination for focus of infection in the upper respiratory tract. His diagnosis was chronic multiple infectious arthritis. We found chronic tonsillitis, adenoids, plus bilateral maxillary sinusitis. A hemolytic streptococcus was secured from both the maxillary sinuses and from the tonsils which, when injected into a rabbit, caused arthritis. The tonsils and adenoids were removed and the diseased maxillary sinuses treated by simple irrigation with argyrol. The sinus disease apparently disappeared but, while the arthritis was improved, it persisted. Six months later an opening was made in each maxillary sinus through the antromental wall and the sinuses treated. The sinus disease disappeared and with it the acute joint trouble. Two years after this, on July 6th last, the mother of the little girl brought her into the pediatric service. The mother stated that since the little girl left the hospital two years ago she has been much improved; the first year she had apparently perfect health, but last winter, following a severe cold, she had considerable nasal discharge. There was no trouble with arthritis at this time; two weeks ago she developed a stiff and swollen left ankle. Examination revealed pus in each maxillary sinus. A small opening was made in the antromental wall and the maxillary sinuses irrigated; vaccines were used with an apparent disappearance of the maxillary sinus disease and of the acute manifestations of the arthritis. Several weeks ago I saw this child. She is perfectly well. She can dance and play as well as any child.

Naturally, when her sinus disease was being treated Dr. Steindler also gave the proper orthopedic treatment to the joint.

This case illustrates two things. First, that paranasal sinus disease may cause systemic infection at the age of three.

Secondly, that paranasal sinus disease in infants and young children is sometimes not easy to permanently cure.

D. M., now nine years of age, is one of our most instructive cases. A brief resume of the case is as follows: When she was four years of age I removed her tonsils and adenoids with much improvement. That was in 1916, when we were not examining our four-year-old children for paranasal sinus disease. The little friend of ours would be today much better off if in 1916 we had examined, diagnosed and treated the paranasal sinus disease which was undoubtedly present. When seven years of age we diagnosed a bilateral sphenoiditis and posterior ethmoiditis. The diseased condition was eradicated. The operation was followed by the most striking improvement in the arthritis, temperature and general condition of the case. She was discharged in December, 1919. In July, 1921, she returned with a recurrence of the acute arthritis. The sphenoid and ethmoid regions, much to my surprise, were perfectly healed and clean.

A careful examination showed pus in the left maxillary sinus. This sinus was drained, and in three weeks there was a most noticeable change in our little patient. From a morose, disconsolate child, suffering from acute pain, she was changed into a happy child, free from pain. Her general appearance was wonderfully improved.

For six weeks before this little patient came into our service she received the most careful metabolic treatment and vaccines in the pediatric service without improvement of her acute trouble. The only thing that gave this little girl relief from her acute symptoms when she was six, and when she was nine, was the treatment of her paranasal sinuses.

R. A., age four, suffered from infectious arthritis involving the joints of fingers and toes. The joints were much swollen and painful. She could not walk. There was as yet no permanent deformity. When I saw her first she had bad tonsils and adenoids, plus maxillary sinusitis. Tonsils and adenoids were removed. Three months later she was much improved, but still suffered with acute attacks of arthritis, with pain and fever. The nasal sinuses were treated for one month. At the end of that time a streptococcus viridans persisted in each maxillary sinus and the arthritis remained active.

Then a small antromental opening was made and the sinuses treated. This was followed by relief of the arthritis. Two weeks ago—that is, sixteen months after she left our service—a report stated that she had been and was perfectly well.

W. F. is a case of recurrence of multiple infectious arthritis that recently returned to our service from Dr. Byfield, professor of pediatrics, with the following note:

"MY DEAR DR. DEAN:

"On of the boys in the arthritis deformans series has been absolutely free from trouble since the operation, which took place about two years ago, I believe. Just a few weeks ago he suddenly had a recurrence. The symptoms were pain the joints with some swelling and stiffness, also some headache and an interesting change in disposition, such as I would suspect would take place in sinus infection. I recall in this case that it was rather difficult to spot the infected area, but it was finally located, and the relief to the boy was the result. This boy has been unusually well since the operation, has had an excellent appetite and has done well in school. In a few weeks since the beginning of the recurrence he has lost weight and the appetite has been poor. The mother seems to feel that the treatment which you gave him was the thing which was responsible for his relatively long period of freedom from trouble, and she is willing to have you go ahead again and not have any orthopedic treatment until the focal infection has been studied.

A. H. BYFIELD."

W. F. is now fourteen years of age. He came into our service at the age of eleven. His tonsils and adenoids were removed July 26, 1918. On September 25, 1918, because of the continuance of his acute joint infection a second examination of his nose was made. A deflected septum with suppurative ethmoiditis was found. The examination of maxillary sinuses was negative. A submucous operation was performed on the septum and treatment instituted. At the end of two months' treatment a hemolytic streptococcus persisted in the posterior ethmoids and sphenoid of each side. These sinuses were very necrotic, and on November 7th they were exenterated. With the apparent disappearance of the sinus trouble the acute arthritis disappeared and returned only after a period of two years. Up to two weeks before the admission

on June 29, 1921, he had been free from trouble. At that time knee and ankle joints become painful and swollen. Preceding this trouble he had had, as he said, a cold and a nasal discharge persisted.

The X-ray examination of the frontals and maxillary sinuses showed them to be quite clear. With the nasopharyngoscope pus was found in each posterior ethmoid and sphenoid region. The left sphenoid was particularly badly diseased. The upper posterior sinuses were again operated and postoperative treatment was instituted. As the result, the nasal discharge is rapidly disappearing and the joints are becoming rapidly better. This case illustrates nicely the fact that when the removal of tonsils and adenoids in a child does not remove the focus of infection that the paranasal sinuses should be carefully examined. It also brings to my mind again, forcibly, the fact that in some cases a cure of paranasal sinus disease in children is very difficult.

Bronchitis is a very common result of paranasal sinus disease in infants and young children. In short, I think that every infant that has bronchitis, even if adenoids are present, should be suspected of having paranasal sinus disease, and the paranasal sinuses should be examined.

Paranasal sinus disease may be the source of infection for pneumonia.

Baby J. S., three and one-half months of age, was under our care for suppuration of the paranasal sinuses plus mastoid infection. Each mastoid had been operated upon, and the mastoid wounds were perfectly clean but not yet healed when the baby developed pneumonia. At the time we were treating the paranasal sinus disease. The baby died on February 1, 1922, and the autopsy is as follows:

The right lung is crepitant throughout, but shows no area of consolidation. The left lung is covered by a thick layer of fibropurulent material. The lung is airless, firm but of a dark color. On cut sections the entire organ is of a deep red color, firm and fairly dry. There is a small amount of free fluid which can be squeezed out of the bronchioles. The whole lung presents a fairly typical picture of bronchopneumonia.

Nasal Cavity.—The nasal cavity was opened by a lateral incision through the base of the skull, which permitted the halves of the skull to be quite widely separated. The walls of the entire nasal cavity were bathed in pus. Smears were made from this pus, and microscopic examination revealed many pneumococci. The pus was particularly thick and abundant in the superior meatus. A probe pushed backward through the superior meatus on either side entered a cavity approximately 6 mm. in diameter, which lay partially within the body of the sphenoid and was filled with thick creamy pus. The superior turbinates were cut away and the posterior ethmoids exposed. These cells were also filled with pus. The middle and inferior turbinates were then removed and the lateral walls of the maxillary sinuses exposed and removed. The sinuses were about the size of a navy bean and were filled with pus, which welled up when the wall was removed as if it were under pressure. There was no macroscopic evidence of ulceration of the mucosa or osteomyelitis. The mucosa of the sinuses was pale and edematous. Dr. Armstrong autopsied the head. Dr. Medlar, head of the department of the pathology and bacteriology of the University of Iowa, was present and both he and Dr. Armstrong expressed the opinion that the source of infection for the pneumonia was the paranasal sinus disease.

There is frequently an interdependence between paranasal sinus disease in young children and asthma. The following two letters from Dr. Byfield, head of the department of pediatrics in the University of Iowa, have some bearing on this subject:

“DEAR DOCTOR DEAN:

“You might be interested to know that Orville Schuler, aged eleven years, has been returned to our service for observation. This patient has had asthma for nine years, the attacks coming on at intervals of about a week, previous to his first admission to this service. He received nasal sinus treatment from you, and with the exception of a very mild attack of asthma a day after his return home, he has been free from every evidence of the disease. He has been able to go to school and has lost a lot of his shortness of breath. These statements have been obtained from the patient himself and are, as far as we can tell, reliable.

ALBERT H. BYFIELD.”

"DEAR DR. DEAN:

"We have just heard from the parents of eight-year-old Roy Schulz and twelve-year-old Emmett Martin, in reply to circular letters. Each one of these children had one mild attack of asthma a few days after leaving the hospital and none since that time. This makes the third case in which satisfactory results have been obtained. ALBERT H. BYFIELD."

One of the characteristics of sinus disease in young children is headache, usually frontal, and interpreted by the parents as being due to eye strain. We may have the neuralgic type from involvement of the nasal ganglion. Sometimes the frontal pain is very excruciating. It is interesting to note that occasionally a tuberculous meningitis beginning will cause the same type of pain as an acute frontal empyema. In acute paranasal sinus disease the patient also complains usually of nasal stoppage or a feeling of fullness in the head.

Infants do not complain of headache. Dr. Byfield is of the opinion that a child complains of headache first in the neighborhood of from five to seven years. He reports that in Dr. Still's textbook, in discussing the migraine of childhood, he mentions that at the age of five years is when the first complaint of headache is usually made.

The following case is very interesting: Mrs. S., aged 44, came complaining of right sided headaches, beginning at the age of six years. Frequently attacks of pain in right forehead and right eye. The headaches have always been of this type. Many times a diagnosis of migraine has been made, and she assumed that this was a headache which was to remain with her for life. In examining the paranasal sinuses, when a maxillary sinus was irrigated the patient immediately said that the pressure caused the typical headache. Eradication of a chronic maxillary sinus disease resulted in an immediate and permanent disappearance of a headache which had persisted for thirty-eight years.

In children, fever usually accompanies acute paranasal sinus disease. In infants the fever is usually very high, 103 to 105½, with daily remissions. It is axiomatic, I believe, with pediatricists that in all cases of fever in infants where the cause is not definitely known, the ears should be examined. As I

view it, the examination of the paranasal sinuses is of equal importance. The fact that an acute ear is present does not exclude acute paranasal sinus disease, and the individual who has in his care an infant with an acute suppurative otitis media in which, after myringotomy fever persists, and who does not make an examination of the paranasal sinuses is liable to make a serious mistake. I have seen several cases where mastoidectomy had been recommended in infants where the cause of the fever was infection of the paranasal sinuses, and the fever disappeared with the treatment of the paranasal sinuses.

Recurrent fever in infants and young children without apparent cause is sometimes due to disease of the paranasal sinuses.

Three years ago, I. H., then seven years of age, was referred to us by Dr. Byfield. He gave a history of attacks of sudden elevation of temperature of about 102 without apparent cause. During the attacks of fever the boy was not ill. He seemed slightly indisposed, and the fever was found by the use of the thermometer. This condition had begun at about the age of two. It had been made better by the removal of the tonsils and adenoids one year before, but persisted. Three years ago the paranasal sinus disease which was present was eradicated, and his trouble has completely disappeared.

Periodic vomiting may be associated with disease of the paranasal sinuses.

F. M., six years of age, came into our service suffering from attacks of recurring fever with vomiting. These attacks are complicated by acidosis. The eradication of an ethmoiditis resulted in a disappearance of the attacks of vomiting and fever.

The work on the paranasal sinuses was done almost two years ago. Three months ago a report from the boy stated that he had not had any trouble up to that time.

The following is the report of a case of periodic vomiting that was very carefully studied by us and in which the result was disappointing:

L. D., age 19 years, entered our service with the following history: Since the age of two he has suffered from periodic vomiting. There is one attack every two to four weeks. The internists are of the opinion that at this time there is a decided functional element in the case. The first attack at the age of two and every one since has the following history: Cold in the head followed by dull headache in frontal region and top of the head, vomiting. Chronic suppuration of all the paranasal sinuses except the left frontal was found. All sinuses have been drained. The number of attacks of vomiting have been greatly decreased, but he still has an occasional very severe attack. We cannot help but feel that in the beginning this was periodic vomiting due to paranasal sinus disease, and that later a functional element appeared in the case so that the eradication of the paranasal sinus disease did not result in a complete cure.

A most common complication of paranasal sinus disease, especially in infants and young children, is gastrointestinal disturbance. This is usually so severe that it is thought to be the primary trouble. As to whether this disturbance is the result of the swallowing of the nasal discharge or the result of the elimination of bacterial products through the gastrointestinal tract, I do not know. Dr. Byfield suspects that the milder cases at least are the result of the ingestion of the purulent discharges and that in the more severe cases, just as we may get an acute nephritis from the elimination of bacterial products, we have an acute gastroenteritis.

Dr. Scruton (Laryn., January 22, page 70) says that gastrointestinal troubles in acute otitis media are due to the ingestion of the purulent discharge from the ethmoids. This purulent discharge is also the cause of the purulent otitis. I do not think we can emphasize too strongly this point made by Dr. Scruton. We must not assume that the systemic disturbances of an acute otitis in infants are always the result of the ear lesion, because they are so frequently the result of a lesion of the paranasal sinuses.

Pyelitis may be related to paranasal sinus disease just as it is related to chronic infection of lymphatic tissue in the nasopharynx and oropharynx. In a personal communication, Dr.

Stucky told me that he had several such cases. We have had two where the elimination of the paranasal sinus disease seemed to be the thing which was responsible for the disappearance of the pyelitis.

Acute cervical adenitis in infants and young children may be the result of paranasal sinus disease.

N. N., seven years old, suffered from very extensive acute cervical adenitis with temperature of 105. X-ray showed well developed frontals. Frontals, anterior ethmoidal and maxillary sinuses blurred. Tonsils and adenoids out one year previously. This boy was very ill. Treatment of diseased maxillary sinuses alone resulted in a disappearance of his trouble in one week's time. I have seen an infant one year of age with the same condition, the result of ethmoiditis, relieved by the treatment of the ethmoiditis.

Recently, Renaud, a French physician (International Medical and Surgical Survey, December 21, 88 b., 348) expresses the opinion that the cause of death in infants who are supposed to die from gastrointestinal disturbance is frequently a suppuration of the mastoid. Our experience is that the suppuration of the mastoid in infants is frequently secondary to the paranasal sinus disease.

I trust that I am not infringing on the province of the pediatricist when I express the opinion that if the paranasal sinuses and mastoid cells are carefully autopsied in all infants who died that a surprisingly large number of acute suppurative infections will be found present in these cavities.

Certainly paranasal sinus disease is frequently the source of infection for acute otitis media. In the infant ward this winter we had an epidemic of acute suppurative otitis media. Every one of the children with infected ears had an acute infection of the paranasal sinuses. When drainage of the ear did not relieve the high temperature, treatment of the paranasal sinuses frequently did. At least four infants died, and in each of the four there was pus under pressure in the paranasal sinuses.

In the obstetric service we had a very interesting case. Baby C., seven days old. At the time of birth the mother had an acute paranasal sinus disease with an acute ear. We secured

a hemolytic streptococci from both paranasal sinuses and ear. When seven days old the baby developed fever. Examination showed suppurative ethmoids, acute otitis media bilateral. Both nose and ears were treated. Later a mastoidectomy on each side was performed. The baby gradually failed, and when two months old died. Autopsy performed by Dr. Medlar, head of the department of pathology in the university. Findings: Little pus in each tympanum; ethmoid and maxillary sinuses filled with thick pus under pressure. Cause of death, paranasal sinusitis and suppurative otitis.

Paranasal sinus disease may cause in children, but not in infants, brain abscess.

Paranasal sinus disease may cause meningitis at any age.

Dr. A. H. Andrews of Chicago (Ill. Med. Journ., Nov., 1921) reports a child six years of age dead from meningitis. At autopsy the meninges were found involved from necrosis of the roof in the sphenoids. The sphenoid was filled with pus and granulation tissue. No examination of the nose had been made before death.

The ocular complications of paranasal sinus disease in infants and young children are most important. There is frequently an interdependence between the infection of the sinuses and phlyctenular conjunctivitis.

Turner, in discussing the etiology of phlyctenular conjunctivitis (Amer. J. of Oph., February, '19), is of the opinion that the ethmoids are the most important etiologic factor in phlyctenular conjunctivitis. He is of the opinion that when improvement of the eyes follows the removal of tonsils and adenoids, it is due to the removal of the latter's influence on the ethmoids, which are present, that results in the improvement of the eyes.

G. B., a colored boy, aged ten years, was referred to us because of phlyctenular conjunctivitis. This boy was in the hospital for six months. When he first entered the hospital a bilateral maxillary sinus which he had was treated and eradicated. The usual local and general treatment for phlyctenular conjunctivitis was given by Dr. Boiler, the ophthalmologist. At the end of his six months of treatment his nose seemed to be perfectly clean, but the corneæ remained injected and photophobia was marked. Dr. Boiler reported that the eyes had

remained absolutely stationary for six months. The paranasal sinuses were cultured, and from the posterior ethmoids and sphenoids on each side a streptococcus hemolyticus was found. The posterior ethmoids and sphenoids were drained. Seven days after the drainage of the posterior sphenoids and the ethmoids the eyes were very much better, and in three weeks all acute trouble and photophobia had disappeared.

L. G., a little child, nineteen months of age, suffered from phlyctenular conjunctivitis, which disappeared with the disappearance of the paranasal sinus disease and reappeared when the nasal trouble reappeared.

E. L., a boy of two and one-half years of age, suffered from phlyctenular conjunctivitis. This boy had bone tuberculosis and tuberculosis of the pharyngeal tonsil plus a nontuberculous maxillary sinus disease. The etiologic factor in the paranasal sinus disease was pneumococcus, type 4. The treatment of maxillary sinuses and the injection of a vaccine prepared from the sinus discharge was of much benefit to the phlyctenular conjunctivitis. This case is interesting because the treatment of paranasal sinus disease in a patient manifestly suffering from tuberculosis was an aid in the treatment of phlyctenular conjunctivitis.

Retrobulbar neuritis is an occasional complication of paranasal sinus disease in children.

Dr. McCullough (Trans. N. Y. Acad. Med., October 26, '21) reports that in one year he saw three cases of retrobulbar neuritis secondary to paranasal sinus disease in children under fourteen years of age.

Dr. J. B. Gregg recently sent me the report of a case of retrobulbar neuritis in a child that was eradicated by the treatment of the diseased posterior ethmoid and sphenoid cells.

White (Larynx, August, '21) reports several cases of involvement of the optic nerve in children five years of age and older.

Orbital cellulitis may result from paranasal sinus disease in infants and young children.

Baby B. O., eighteen months of age, had orbital cellulitis, secondary to maxillary sinusitis and ethmoiditis. The orbital wall of the ethmoid was broken down, and the neighboring bones were also involved.

P. B., aged four, had an acute pansinusitis suppurative with orbital cellulitis on the same side. We made an opening in the maxillary sinus. The use of suction, nasal irrigation and argyrol in the nose resulted in the disappearance of the orbital lesion.

Baby H., six months of age, was in our service with a diagnosis of maxillary sinus disease secondary to posterior cleft; suppurative otitis media had developed. While the patient was under treatment for mastoid involvement an orbital cellulitis with abscess of the cheek formed. It is interesting to note that in this six-months-old baby the existence of the maxillary sinus had been proven before the appearance of the orbital cellulitis.

XXII.

OBSERVATIONS ON THE ART AND TECHNIC OF BRONCHOSCOPY AND ESOPHAGOSCOPY.

BY THOMAS HUBBARD, M. D.,

TOLEDO.

The voluminous publications of this subject rather deter one from attempting to interest those who have encountered and solved most of the problems connected with the diagnosis and operation for removal of foreign bodies from the lower air passages and esophagus. But the very nature of this work—the variety, the pathologic complications and the emergency features—gives each operator a different angle of observation, and personal experiences and conclusions aid in establishing sound methods.

Under the heading "method" one naturally thinks first of anesthesia. This refers, of course, to general anesthesia. This should be decided in accordance with the conception of the term "deliberate surgery." In foreign body bronchoscopy one undertakes a surgical operation of undetermined and perhaps extreme difficulty. It is easy to scare up arguments against ether anesthesia in all kinds of operations. Some are valid and some are not. Occasionally the risk is forced. One cannot generalize. Postulates express the individual preference too positively. Each case is a study and I desire merely to convey the conviction that prohibition of anesthesia is a handicap and tends to establish an impractical standard. But I would qualify endorsement of general anesthesia, when indicated, by the statement that the anesthetist must be one prepared to aid in the emergency disturbances incident to the operation. He must know the danger signs, warn the surgeon timely and know the restorative indications. The oxygen outfit is essential for this purpose. Posture is important and he should be experienced in this matter.

Supposing that there be dyspnea in the case presenting for bronchoscopy. First determine the location of the obstruction: Laryngeal, foreign body in the trachea or bronchus, pneumo-

nia, or a flooded lung. In so far as laryngitis is concerned, it is my experience that ether anesthesia will relieve a spasmodic, croupy dyspnea, and I would add that in the hands of the average operator there is less danger of laryngeal trauma in passing the bronchoscope in the quiet, anesthetized patient. Several of the cases in this collection had laryngeal dyspnea prior to operation, but in none has there been serious aggravation by operation. Fortunately emesis is physiologically inhibited by intubation of the larynx. No emesis with the open glottis. This makes the no anesthesia method possible, but does not condemn anesthesia. The question of tracheotomy in laryngeal dyspnea will be considered later.

A tracheal or bronchial dyspnea is not a contraindication to ether (plus oxygen if needed), and I would say the same of even bronchial flooding often misnamed pneumonia. It is in precisely the type of case having dyspnea that deliberate surgery is essential. The cause must be removed and the operator should feel at liberty to select that method which gives him the best chance of successful operation.

Sudden asphyxiation is usually due to one of three causes: Either it is shifting of the foreign body blocking the trachea or glottis, or it may slip over into the open bronchus, or it may be due to the release of pent up secretions of the invaded bronchial tract overflowing into the functioning lung. In the first instance prompt insertion of the tube will displace it into the trachea, giving temporary relief, or it may be grasped in the larynx. In the other emergencies one must depend on aspiration and removal of the foreign body.

This complication, sudden asphyxia, occurring before or after removal of the foreign body, has caused fatality on the table, and it is probably the basis of prejudice against general anesthesia. But as we get experience and study all of the factors the apparent objections to anesthesia are less convincing. In my experience the aspirator is the life saver.

This matter of bronchial flooding in foreign body cases introduces a question of pathology. We are in the habit of labeling a case pneumonia or bronchopneumonia when there is percussion dullness, typical rales or diminished or no respiratory sounds, limited thoracic expansion, confirmed may be by fluoroscope, but we should bear in mind that there is often

absence of evidence of a real infection inflammation. There may be considerable fever, due perhaps to the retained secretions. In peanut aspiration there are fragments, mouth contaminated and irritating, which produce excessive secretion in bronchi and bronchioles, but there is another factor in cases having actual plugging of a main or secondary bronchus. Intrapulmonary air pressure is normally minus 1 or 2 mm. Hg. inspiratory and plus 2 to 5 mm. Hg. expiratory in quiet respiration. In forced respiration, as in dyspnea from obstructed air passage, the inspiratory negative pressure may be as high as minus 57 mm. Hg. and in expiration plus 80 mm. Hg. This extreme range of pressure from the normal tends to engorge the delicate vascular mucosa of bronchi and bronchioles and alveoli. The still higher positive expiratory pressure would tend to unload the congested vessels, but in dyspnea due to a plugged bronchus we have an almost continuous high negative pressure below the point of obstruction of one pound (50 mm. Hg. = 2 inches Hg.), more or less, varying with the degree of obstruction and vigor of the subject. (Two pounds' negative pressure will cause decided hyperemia of the nasal mucosa, four pounds will draw blood and that, too, in tissues more tolerant of high negative pressure.) Irritation hyperemia plus negative pressure seems to account for the rapid flooding of a bronchial tract below the point of obstruction. Limited thoracic expansion is one of the cardinal symptoms of obstructed bronchus. There is evidence that this is due in part to physiologic inhibition, but it is also an indication that the air content is displaced by fluid. But this is not a true pneumonia nor an inflammatory edema, as has been made clear by McCrae, Spencer and Jackson. The proof of this is found in the fact that in many of these cases the removal of the foreign body and deep aspiration will promptly restore lung function, and within a few hours respiration becomes quite normal and the fever vanishes. Retained secretion is a foreign protein and causes the rise of temperature. A brief report of three cases will make clear the important points and the operative indications.

A baby of sixteen months had a fragment of peanut in the right bronchus for seven days. The physical examination, malaise, temperature and coughing warranted the usual diag-

nosis of bronchopneumonia. The infant nearly passed away from the flooding of the open bronchus after removal of the foreign body. With the tube in situ the mucus could be seen to well up in the tube until it reached the level of the breathing fenestra, and as it overflowed into the left it caused asphyxia and cyanosis. The pulse remained normal. Prompt removal of the tube, which was filled with the thick, tenacious mucus, turning the infant to the right side and then upending it, caused the overflow to come out of the mouth. Aspiration would have been better, but we tried the posture method in this case and it was effective. This patient had no fever after operation and recovered so promptly that the diagnosis of bronchopneumonia was not confirmed.

Another case, that of a boy of three and a half years, was sent to Toledo Hospital with a diagnosis of "arachidic bronchitis" of four days' duration. The fever ranged between 102 and 104 degrees. The radiographic plates showed the left lung to be almost airless, limited expansion of the left thorax, no excursion of the left diaphragm observed by fluoroscope, no respiratory sounds in the left thorax, respiration very rapid and the patient quite exhausted. Two peanut fragments were removed from the lower left bronchus, Dr. Galbraith operating. Probably they plugged both secondary bronchi, which accounted for complete obstruction. In this case the overflow of mucus was prevented by aspiration. The contents of the wash bottle illustrate the importance of thorough deep aspiration. (See lantern slide.) Small pieces of peanut and debris in a large amount, an ounce or two, of thick, tenacious, somewhat purulent mucus accounted for the findings of the physical examination and the high temperature. Retained secretions caused the fever. As an additional precaution this patient was turned on to the affected side even after aspiration. There was prompt relief of coughing and no fever after operation. An hour later air was entering this lung almost normally.

Metallic foreign bodies blocking one or more secondary bronchi will produce about the same degree of fever and the same symptoms of pneumonia. A brass pencil cap lodged low down in the left bronchus gave a clinical picture very similar to the last mentioned case, and the symptoms of broncho-

pneumonia vanished as soon as the pent up secretions were withdrawn after removal of the foreign body.

(These three cases are not included in the group of specimens.)

It is evident that infection is in such cases of slight degree and that the seromucus is chiefly due to irritation and the almost continuous high negative pressure below the point of complete obstruction. The inference is that the term broncho-pneumonia overstates the pathologic condition. It is more of the nature of a negative pressure bronchorrhea. "Arachidic bronchitis" implies a type peculiar to peanut aspiration and undoubtedly in this accident irritating, mouth contaminated debris, is liable to enter small bronchioles, but it is probable that any foreign body that plugs the main bronchus or one or more secondary bronchi will cause about the same symptoms.

The surgical lesson is that the operation for removal of a foreign body from a bronchus is not complete until the debris and pent up secretions are evacuated as thoroughly as possible, and as a precaution against overflow into the open bronchus the patient should be turned on to the affected side.

Now a few words concerning tracheotomy. The lantern slides and legends show the contrast between prebronchoscopic days, the early period of bronchoscopy, and present day practice. The real indications for tracheotomy are either of emergency type or to aid in certain extraction problems. For instance, in laryngeal impaction tracheotomy may be forced. An extreme degree of laryngeal obstruction will cause anxiety, even after extraction of the foreign body which has caused it, and rather than run the risk of being forced to do it in a hurry it is well to get a cannula in promptly. I have had no experience with postoperative intubation, but an inflamed larynx does not tolerate a tube well, and extubation is liable to be delayed—a rather serious complication. As stated, none of the cases in this series showed postoperative evidence of laryngeal trauma.

In foreign body lung abscess tracheotomy is indicated to aid in the aspiration of purulent contents. It is a nasty job to work showered with foul pus, and this can be avoided by a few days of frequent aspirations prior to exploration by the bronchoscope.

The lower route aids in reaching and exploring the orifice of the upper right lobe bronchus for removal of a foreign body, the angle of approach being more favorable than by the upper direct method, and also the shorter tube makes easier the mechanical manipulations in complicated conditions conducted, if necessary, under the guidance of the fluoroscope.

There is an interesting phenomenon connected with this matter of prying around in the trachea and in the bronchus. I have observed syncope not due to asphyxiation, a sudden slowing of the pulse rate. In one case my associate, Dr. Galbraith, was searching the lower left bronchus for some more peanut fragments, two already extracted. The pulse dropped, as the anesthetist estimated, to less than 30, and it promptly came back to normal when the extension tube was adapted to the proper axis. Pressure on or tension of the vagus filaments to the cardiac plexus may be the cause of inhibition. It is the duty of the anesthetist to watch for such disturbances and warn the operator timely. Probably some fatalities of this type are ascribed to status lymphaticus.

The nature of esophagoscopy is similar to bronchoscopy, but the anatomic construction and frequent impaction complicate and add a factor of trauma. Above all this, work must be deliberate, and to that end general anesthesia is indicated, excepting, of course, the obviously easy ones. Untimely emesis may become a real complication. Dr. Jackson has delineated the complicated mechanical manipulations necessary, and certainly one must have the aid of complete relaxation and forget the time limit to do this kind of work deliberately.

Posture is a matter of great importance. In adults one can get a suggestion of the esophageal axis by examination of the dorsal vertebral curve. I think that the operator can keep his sense of orientation better by the supine horizontal posture as the standard, varying slightly to keep exactly in the axis of the tube explored.

A few words about open safety pins in the esophagus: The most popular type is made of brass, and fortunately this is less dangerous than the plated steel pin, as it is less sharp and more flexible. In this group fifteen cases presented the hook method of reversing the point was used. Only one was extracted by

forceps, and that is the only case in which there was trauma. This child had a throat infection on the way, having traveled far, and the trauma of extraction aggravated the cervical adenitis and general cellulitis. In this case the guard was imperfectly attached and was pulled off by the hook, leaving the pin crosswise until it was grasped by forceps and extracted.

It should be added that one must study the plate measurements to determine if turning the open pin is practical, and if not the shield hook method, covering the point and pulling it into the beveled lip of the tube, or the method Dr. Jackson now advises should be resorted to; that is, grasping the point and shielding it in the beveled tube end.

This special art has enabled the trained vision to penetrate deeply into the naturally obscure anatomic regions to meet surgical indications with precision and study with accuracy hidden pathology. I should like to put into words sentiments of appreciation of the work of the founders of this surgical art. Endoscopy is peculiarly spectacular, and it is high tension work with startling end results. Following the moment of the exaltation over a surgical achievement there comes to mind the men who have written this chapter in the history of laryngology. Joseph O'Dwyer developed the art of intubation of the larynx, and Gustav Killian and Bruenings perfected tubes and proximal illumination. Ingals and Coolidge imparted the inspiration to their colleagues. Jackson has blazed a new way in the perfection of the technic and instrumentation and established standards that will keep the next generation alert. Mosher, Lynch, Arrowsmith and Lynah have done foundation work in special lines. Many others have contributed to perfect detail. We honor them when we realize on their ideals.

XXIII.

THE RADICAL FRONTAL SINUS OPERATION, WITH REPORT OF CASES.

BY FIELDING O. LEWIS, M. D.,

PHILADELPHIA.

In a large percentage of cases in which there is a chronic, suppurative process of the frontal sinus, the radical operation affords the best means of a permanent relief from such distressing symptoms as recurring or persistent pain and headaches, purulent nasal and postnasal discharge, alarming vertigo, gastrointestinal lesions and focal infections. There is a certain group of cases in which some form of radical procedure is unquestionably the only method of treatment. I would mention in this connection those cases with external fistulae, intracranial complications, extremely large sinuses, with their often present septal divisions, bone necrosis, severe orbital complications, and when the intra-nasal operation has been unsuccessful.

By radical operation is meant the Killian, or some of its many slight modifications. This type of operation affords the surgeon the opportunity of carrying out certain surgical principles which are essential to success in these cases. By the Killian method of approaching the sinus, it is possible to visualize the entire field of operation, which is the only definite way of determining what is within the sinus. If foreign bodies are present, as in one of my cases, a piece of drainage tube was removed; if bone cysts have formed, three of which were found in another case; if anatomic variations (which is the rule rather than the exception) are encountered; if the mucous membrans of the sinus is hyperplastic, with its ever present polypoid formation; if there is necrosis of the posterior plate—a thorough house cleaning is, to my mind, clearly indicated. This cannot be accomplished by chiseling a small hole through the anterior plate, any more successfully than a mastoid operation can properly be performed through a small opening in the mastoid cortex. The intranasal operation would be about

as useful as a needle puncture for the cure of chronic suppurative maxillary disease.

In my somewhat limited experience in about forty cases operated on radically, a complete removal of mucous membrane, a careful examination of the septa when present, as diseased cells are often concealed at their base, and good nasal drainage, are imperative in order to secure a complete cure. Careful postoperative treatment is always a feature often neglected. The case should be watched, dressed and treated by the surgeon or his assistant who is fully familiar with these cases.

While some writers mention the decline in popularity of the radical operation in favor of the intranasal route, I wonder if the intranasal operation, even in suitable cases, has been all that its advocates have desired. In my own experience, with one or two exceptions, it has accomplished no more than that secured by intranasal drainage through removal of hypertrophies and septal deviations.

A postoperative deformity depends, of course, upon the size of the sinus and the amount of necrosis of the anterior plate. With such brilliant results being now obtained by plastic surgery, marked deformities should be readily corrected by the transplantation of bone, cartilage or fat. I have found that the patients, particularly those past the age of vanity, are so delighted with the cessation of the symptoms, that they prefer to retain their defect, though sometimes marked, rather than to submit to what some have feared might be a return of the old trouble.

From a series of some forty cases I have selected a few, which to me have presented interesting pathological and surgical problems.

Case 1.—On account of the unusual symptoms and extensive involvement of all of the sinuses, this case is reported more or less in detail. W. B., male, aged 54 years; married. Occupation, house painter for the past 25 years. Was admitted to the medical ward of Jefferson Hospital in December, 1921, suffering with bronchial asthma. Chief complaint: Shortness of breath. Family history: Nothing of interest known. Personal history: Used alcohol and tobacco moderately. Had typhoid fever in 1879; pneumonia in 1890. Denies venereal disease.

Present illness: In January, 1911, patient had his first attack of asthma. Was engaged in inside painting when first attack occurred, which lasted two days and he was away from work one week. Mild attacks of short duration followed at various intervals for seven years. Three years ago he had another severe attack; since that time the asthmatic attacks have come on at irregular intervals in close sequence and at all times of the year. There seems to be no seasonable influence. For three years he has received autogenous vaccines. The vaccines exaggerated the attacks.

Condition on admission: A poorly nourished adult, white male. Marked gastric symptoms, and breathing so difficult that he was unable to lie down.

Physical examination: Eyes and ears normal. There is a large septal perforation following a submucous resection some years ago. There are nasal polypi in both ethmoidal regions and evidence of ethmoidal exenteration. Practically no nasal discharge visible. Marked evidence of oral sepsis from diseased teeth. Thorax, abdomen and extremities negative. Urine, faint trace of albumin, occasional hyaline and granular cast. Complete blood count shows 18,800 white cells and slight variation in size, shape and stain of red blood cells. Sputum on numerous occasions negative of tubercle bacilli. Many gram positive diplococci. Gram positive streptococci and staphylococci. Blood, Wassermann negative. Patient's attacks are relieved by the administration of hypodermic injections of adrenalin, chloride and nitroglycerine. Nitrate of amyl pearls and large doses of morphin were given on various occasions. Rigid diet was also instituted. While in the medical ward the progress had been very unsatisfactory. Injections of dust extractions gave no relief, and the patient had often as many as six asthmatic attacks in twenty-four hours, which became more severe in character. So severe were the attacks that inhalations of ether were administered, as large doses of adrenalin chloride and half grain doses of morphin were of no avail.

Following an examination of the nose and throat, the patient was transferred to the laryngologic service. X-ray studies by Dr. Manges showed considerable cloudiness of both frontal, both ethmoidal and maxillary sinuses.

Operation: December 31, 1921, under intratracheal anesthesia, the right frontal sinus was first exposed by the Killian method. The sinus was found quite large, and filled with a peculiar mucoid substance. There was necrosis of the anterior plate, and the septum between the frontal sinuses was necrotic. The left frontal sinus was then opened and the dura was found exposed by necrosis of the posterior plate for an area the size of a twenty-five cent piece. Both maxillary sinuses were opened and found equally diseased, with marked necrosis of the outer wall and the nasosinusal wall. The remaining ethmoidal cells were found diseased and exenterated. The sphenoidal sinuses were also found to contain pus. From the amount of bony necrosis of the entire skull, and from the character of the discharge, tertiary lues was suspected. Though a negative spinal fluid examination was found, the patient was given neosalvarsan. Almost immediately following the extensive operation on all the sinuses and the administration of the neosalvarsan, the patient showed marked improvement, which continued until his discharge on February 14. While he has some attacks of asthma, they have been far less severe and much less frequent.

Case 2.—J. F. F., male, age 31; married. Occupation, postal clerk. Patient was seen in consultation on July 2, 1921. Chief complaint: Pain in right frontal region. Some nasal discharge. Symptoms of constant cold and epistaxis. General health good. Family history: Father died of carcinoma; mother dying of the same disease. Personal history: No severe illness until the present time. One child living and well.

Examination: The right nostril was filled with a freely movable, hemorrhagic growth about the size of a large white grape, attached to the middle turbinate. Bleeds freely when touched. On transillumination, the sinuses were unusually clear. X-ray examination negative. Under local anesthesia the growth was removed with a portion of the middle turbinate and sent to the laboratory for examination, report of which was given as an epithelioma, following which 50 milligrams of radium was placed in the nose and crossfired with plaques on the outside. This was repeated in the course of six weeks.

Patient returned in December, 1921, with a return of the growth and suffering with intense headaches on the right side.

The growth was again removed and radium introduced as before. X-ray made at this time shows a normal condition of the antrum on both sides, which was clear without evidence of pathology. The sphenoid sinuses showed clearly on each side, with no increased density. The frontal sinuses are also clear.

The patient was seen again in the course of a month. The nostril was free of any evidence of growth, but the patient's headaches had increased to such an extent that he was unable to sleep or attend properly to his occupation. He had occasional attacks of vomiting. His general condition gradually grew worse and was admitted to the Jefferson Hospital for further observation. Further X-ray studies were made, and the right frontal sinus was found cloudy, the other sinuses were clear. No pus could be seen in the nose. He was apathetic. Pain was so intense that morphin had little effect. Vomiting became worse, with no rise of temperature. Neurologic examination revealed all reflexes normal; the eye grounds were negative as to intracranial pressure. Wasserman was negative. Lumbar puncture showed no increased pressure and the spinal fluid normal.

Operation: On account of the extreme headaches and X-ray findings, the frontal sinus was opened externally by the Killian method, and the sinus was found to be filled with an apparent coagulation necrosis of the mucous membrane. The material of the consistency of gelatine, was removed en masse, leaving the bony wall clean and shiny. There was no evidence of any bone involvement. The sinus was treated in the usual manner. The patient's symptoms were not relieved, and death occurred at the end of the first week. A bacteriologic examination of the material was sterile. Postmortem examination was refused.

Remarks: It at once occurred to me whether it was possible for the radium to have had such effect upon the mucous membrane of the sinus, and if so, did the radium affect the brain cells?

Case 3.—A. R. H., male; married; aged 43 years. Occupation, bookkeeper. Admitted to the Jefferson Hospital, Philadelphia, September 21, 1910.

Personal history: Had scarlet fever and diphtheria when a child. Has had more or less discharge from his nose since childhood. Nasal polypi were removed by his family physician a month before his admission.

Present illness: Chronic, yellow, foul and profuse discharge from the left nostril. For the past few weeks has had severe pain and some fever.

Condition on admission: General physical examination negative. The left eye was completely closed by a swollen upper eyelid. Extreme redness and marked tenderness. Photophobia and marked lacrimation. Examination of the nose showed both nostrils filled with nasal polypi and a mucopurulent discharge. X-ray showed involvement of the left frontal and both ethmoidal sinuses.

Operation: Nasal polypi were first removed under local anesthesia. The frontal sinus was then opened by the Killian method, under general anesthesia. The entire anterior plate was necrotic and the sinus was filled with pus and polypoid mucous membrane. The packing was introduced with external drainage and the patient made an uneventful recovery.

Three years later, the patient returned with a swelling in the same location, but an absence of redness and tenderness. On reopening along the line of the former incision it was found to be a mucöcele, which was thoroughly curetted, packing again introduced, and the patient has had no further trouble up to the present day.

Case 4.—Dr. W. W. W., male; married; aged 33 years. Admitted to Jefferson Hospital October 15, 1921. Family history negative.

Personal history: Patient had a gastroenterotomy during his college course, for gastric ulcer. Has had a purulent discharge from the nose following repeated attacks of cold, while a student. Both maxillary sinuses were operated on radically, also exenteration of the ethmoids on both sides.

Present illness: Patient complains of severe headaches, more pronounced when the nasal discharge is scant. Attacks of loss of consciousness, epileptiform in character. Loss of weight and inability to practice his profession.

Condition on admission: Polyps were found in both nostrils in the region of the ethmoids. Mucopurulent discharge

more pronounced in the left side. Transillumination of the sinuses shows a darkened area over the frontal region. X-ray showed marked involvement of the left frontal sinus, cloudiness of the right. Blood Wassermann negative.

Operation: On November 15, 1921, under intratracheal anesthesia, a double Killian operation was performed. Both sinuses were extremely large and deep, with several septal divisions. In the left frontal sinus were found three bone cysts, the largest being about the size of a robin's egg. The other two were somewhat smaller. These contained an amber-colored fluid. There was no necrosis of the posterior plate.

Subsequent history: When last seen, a few weeks ago, the patient had gained twenty pounds in weight, was attending to his daily country practice, and has had no further attacks of epileptiform seizures and no nasal or postnasal discharge.

Case 5.—L. B., female, single; aged 59 years. Admitted to Jefferson Hospital January 29, 1920. Family history negative.

Personal history: Some two years previous had an attack of influenza. This was followed by a purulent nasal discharge and swelling in the region of the left frontal sinus. An external opening in the frontal region was made by a surgeon, and the sinus curetted. Has had a constant discharge from an external sinus in this region almost continuously, with a purulent discharge from the nose.

Present illness: Complains of severe headaches, discharge from an external fistula above the left eye, and purulent discharge from the nose, and frequent colds.

Examination shows a fistula extending into the frontal sinus, some polypi, and a mucopurulent discharge in the left nostril. X-ray report shows slight cloudiness of the left ethmoid cells and to a less degree of the right. Left frontal sinus apparently obliterated by a former operation. Maxillary sinuses normal. Blood Wassermann negative. General physical examination negative.

Operation: Under intratracheal anesthesia the sinus was exposed along the line of the previous incision. The sinus was found to be filled with pus and granulations. The wound was packed in the usual manner.

Subsequent history: A few months following the operation the patient had some discharge in the region of the old fistula and marked deformity at the inner angle of the wound. Under anesthesia the fistula was again curetted and packed and the deformity corrected by a plastic operation. For the past year there has been no evidence of any discharge of further symptoms.

Case 6.—C. B., male; married; aged 41 years. Occupation, traveling salesman. Admitted to Jefferson Hospital March 7, 1920. Family history negative.

Personal history: For the past five or six years the patient has had severe headaches which were diagnosticated and treated as migraine. Had consulted many physicians, and relief could only be obtained by hypodermic administration of morphine. Blood Wassermann had been made repeatedly and always found negative. No X-ray examination had ever been made up to the time of his admission.

Condition on admission: No tenderness or swelling in the region of the frontal sinus could be obtained, and no purulent discharge could be found in the nostrils. Transillumination showed a darkened area in the region of the right frontal sinus. X-ray showed a rather large sinus and extremely cloudy. The other sinuses were apparently clear. General physical examination negative.

Operation: Under intratracheal anesthesia the frontal sinus was exposed in the usual manner and found to be filled with thick, creamy pus. The mucous membrane was thoroughly removed and free nasal drainage instituted. As the ethmoids did not seem to be involved they were not disturbed. The patient made an uninterrupted recovery.

Subsequent history: Has had no further symptoms referable to his sinus, has had occasional headaches, but less severe than before the operation.

Case 7.—H. D. C., male; married; aged 39 years. Was admitted to Jefferson Hospital June 22, 1921. Family history negative.

Personal history: For several years the patient has complained of recurrent headaches and postnasal discharge. Had consulted physicians repeatedly without relief. Right frontal sinus operated on one year ago. Discharging fistula since.

Was referred by a former patient, who had had a frontal sinus operation.

Condition on admission: General physical examination negative. Wassermann negative. There is a discharging external fistula over right frontal sinus, pus in right nostril. No polypi. X-ray report by Dr. Manges showed marked cloudiness of right frontal sinus. The sinus was considerably larger than the left. The other sinuses were clear.

Operation: Under intratracheal anesthesia the right frontal sinus was exposed by the Killian method and was found divided by four distinct septa, each compartment filled with polypoid degeneration of mucosa and pus. Two external iodoform drains and one intranasal iodoform drain were introduced and the wound closed in the usual manner.

The patient was discharged in ten days, with a cessation of all symptoms.

Case 8.—Mrs. C. J. H., aged 33; married. Admitted to Jefferson Hospital July 13, 1921. Family history: Father died of tuberculosis. Personal history: General health good until October, 1920.

Present illness: Has had repeated colds since March, 1921, complicated by acute bronchitis and some symptoms of asthma. Shortly afterward had severe pains over the left supraorbital region and radiating into the eyeball. Frequent nausea and vomiting. Vertigo for the past week, and spots before the eyes. Thick, foul nasal and postnasal discharge.

Condition on admission: Negative, with the exception of the purulent nasal and postnasal discharge, and hypertrophied tonsils. Wassermann negative. Temperature, pulse and respiration negative, urine clear. X-ray by Dr. Manges: "There is marked cloudiness of the left frontal sinus, the left ethmoid and left maxillary antrum. The frontal sinus is rather large. There is an abscess apparently at a molar tooth, the crown of which has been destroyed, in the left upper jaw. This is rather close to the antrum.

Operation: Under intratracheal anesthesia, the usual Killian incision was made, exposing the left frontal sinus, which was filled with foul pus and granulations. There was also partial destruction of the supraorbital ridge by necrosis. As much of the ridge was left as possible, to avoid a marked de-

formity. The left maxillary sinus was operated on radically by the Denker method, the sinus being filled with pus and polypi. The diseased tooth, which extended into the maxillary sinus, and was no doubt the cause of the infection, was extracted at the same time.

The patient was operated on July 14, 1921, and discharged July 22, 1921, with absence of all previous symptoms.

Case 9.—H. K., male; aged 37 years; married. Occupation, railroad brakeman. Was admitted to Jefferson Hospital November 10, 1921.

Chief complaint: Chronic, profuse, foul nasal discharge and frontal headaches. Family history: Negative. Personal history: Negative.

Present illness: For some months has had intermitten swelling with pain over the left eye, following an acute cold in the head. Feeling of heaviness in the head; some slight dizziness; noises in the right ear, and slight deafness.

Condition on admission: Well nourished man. Profuse, purulent discharge in the left nasal chamber. General physical examination negative. Blood Wassermann negative.

X-ray by Dr. Manges: "I am convinced that the patient has a left frontal sinus, although it is not quite as large as the right one. I feel that this sinus has been occluded for a long time and that there has been considerable bone thickness such as you get in a mastoid, and that the natural, smooth outline of the sinus is destroyed. I believe that you will want to do a radical operation and I would advise that you make a small opening at first, because I am not certain how far down the sclerotic bone extends."

On transillumination, the left maxillary sinus was found cloudy. Paracentesis revealed a large amount of foul-smelling pus.

Operation: Under intratracheal anesthesia, the left frontal sinus was exposed in the usual manner. The sinus was found quite large, contrary to the findings of the X-ray, and when opened, a large quantity of foul-smelling pus gushed out. There was also considerable necrosis of the anterior plate. The left maxillary sinus was operated on radically by the Denker method and found to contain a large amount of pus and poly-

poid tissue. The patient made an uninterrupted recovery and has had no further complaint.

Case 10.—L. C., white, female; aged 10 years.

Chief complaint: Swelling of the right eye, with pain, redness and fever. Nasal and postnasal discharge.

Family history: Mother and father living. Three sisters and three brothers living and well.

Personal history: Had diphtheria when eight years old. Tonsils and adenoids were removed about a year and a half previous to present illness.

Present illness: The patient's mother stated that there had been a nasal and postnasal discharge, purulent in character, for several months past. Five days before the patient consulted the ophthalmologic department of Jefferson Hospital, a swelling was noticed with pain about the eye. No symptoms of this character had previously been noticed. The patient was admitted to the eye department of the Jefferson Hospital February 22, 1921. Under anesthesia the ophthalmologist made an opening into the orbit, thinking the condition was primarily an orbital infection. On introducing a probe, he found that the disease extended into the frontal sinus. The patient was then referred to the nose and throat department.

Examination: Well nourished, female child. The right eye was closed by swelling and marked induration of the upper eyelid. Examination of the nose showed polypi and mucopurulent discharge in the right nostril. X-ray examination reports marked involvement of the right frontal sinus and right ethmoid. Left side clear. Blood Wassermann negative.

Operation: March 1, 1921, under general anesthesia, the usual Killian incision was made over the right frontal sinus and the sinus exposed, which was found filled with pus and granulations and the anterior plate necrotic. The ethmoids were also found diseased and were exenterated. The usual dressings were applied and the patient made a rather prolonged recovery because of a more or less persistent discharge through a fistula, which developed in the incision made into the orbit. She was discharged from the hospital in May, 1921, and has had no further symptoms.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of May 8, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHN, IN THE CHAIR.

SCIENTIFIC PROGRAM.

JOHN P. MINTON, Ph. D., and ROBERT SONNENSCHN, M. D., presented papers entitled:

1. **"Study of Reinforcement of Sound by Means of the Schaefer Resonators."**

See page 45.

2. **"Use of Tuning Fork Stem for Both Air and Bone Conduction in Rinne Test."**

See page 85.

DISCUSSION.

DR. EDWIN MCGINNIS asked if they had tried stuffing cotton in the middle of the tube to see if there was interference with the sound.

DR. SONNENSCHN replied that they had not.

DR. G. W. BOOT asked if they had always used the same length of tube.

DR. SONNENSCHN replied that they had and that it was twenty-six inches long. While it was impossible to demonstrate their point absolutely, it was a fact that the sound waves passed along the lumen of the tube, very little going through the wall and very little being transported by the tube wall itself.

DR. E. L. KENYON asked why they could not use a soft insert in the middle ear instead of the hard rubber tube, perhaps just the tube itself.

DR. SONNENSCHN said they had tried that, but it was hard to hold it in place. The tip being pointed held it nicely in the

ear without striking any of the outside portion of the ear. It was just the size ordinarily used with the Politzer bag.

DR. J. HOLINGER said that there has always been controversy where he could not see any reason for it. In the Rinne test we know that the handle of the tuning fork vibrates as long as the prongs do. It is therefore immaterial whether we observe the handle or the prongs, since it is only a question of time. It was the aim of Bezold to make all the tests as simple as possible. The question of human equation enters into the tests, and the less and simpler apparatus are used the more reliable and the more uniform the results will be. Also, the less the excitement of the patient the better the result will be. It is true, some points are theoretically not explained. In regard to this, Bezold uses the comparison of hearing the heart sounds by means of the stethoscope. It is not known accurately how and where the heart sounds are produced, but it is known that if there are certain changes from the normal sounds we find at the postmortem certain characteristic changes of the valves of the heart (stenosis, etc.). Nobody would go so far as to advise throwing the whole auscultation of the heart overboard because we do not know how the sounds of the heart are produced or how the abnormal sounds can be explained. In his opinion, the only way to attack the question is to say that if there is a negative Rinne in a given case and a middle ear affection is not found at the postmortem, then there is room for an argument. Observe the facts accurately, immaterial of the explanation. Many an explanation was not found because the facts were not observed accurately.

The work of Dr. Sonnenschein was important and showed that he is thinking. Dr. Holinger believes in simplifying, not complicating these difficult matters.

DR. G. E. SHAMBAUGH believed that the work with resonators will bring out some very interesting facts. The method in vogue of testing different parts of the tone scale by such means as tuning forks brought out a good many incidences where gaps in the tone scale existed; that is, where patients were not able to hear the tones over a certain area. By intensifying these tones with resonators many of these apparent gaps in the tone scale disappeared. It seems likely that with enough

amplification there might be found to exist no real tone gaps; that is, areas where one cannot hear a tone, provided the entire function of the labyrinth has been destroyed. It is a practical point of great importance in the education of a child with defective hearing; that is, training the child to speak to ascertain whether there is any remnant of hearing. By use of resonators this may be determined more readily. Even a very small remnant of hearing is often of great assistance in training a child to speak.

The study of the Rinne test is very interesting. Dr. Shambaugh believed that the Rinne test taken by itself gives more definite information than does any other single test in determining whether the cause for the defect is from obstruction in sound conduction or a defect in the sound perceiving apparatus. The Rinne test is perhaps more valuable than all other tests put together in determining this point. In making use of this test one has to take into consideration whether one or both ears are involved. A patient with a one-sided defect that is at all marked is bound to have a Rinne negative, no difference whether this defect is due to obstruction in conduction, fixation of the stapes or whether this defect is due to degeneration in the nervous mechanism.

There seems to be an impression that bone conduction can be determined separately for the two ears. As a rule, it will be found that the bone conduction is approximately the same when the fork is placed over the mastoid process in an ear where we are clinically satisfied that there has been a total destruction of function as when the fork is placed over the mastoid of the opposite normal ear. It is the exceptional case where, under circumstances of this sort, one is able to detect a slight difference in the duration of bone conduction of the two sides.

DR. JOHN P. MINTON (closing), answering Dr. Boot's question, said that the length of time the sound can be heard with the resonator is less than without it. The degrees of bone conduction in old age had nothing to do with resonance, but the change was probably due to partial degeneration of the internal ear mechanism and not at all to other changes that go on.

DR. ROBERT SONNENSCHN (closing) stated that he had the pleasure in 1909 of becoming acquainted with Dr. Schaefer and became interested in his resonators. As he had said in 1912, when he read a previous paper on this subject, the great advantage of the resonators lies in several facts. They were first made use of for experimental purposes by the great Helmholtz. As the resonators were spherical and tested the whole range of sounds from 100 to 1,000 vibrations, it was necessary to have a great many of them, and it consumed much time in shifting from one sphere to the other, besides being very expensive. Koenig in Paris also devised a set and Edelman has a set, but they all have a great many parts. Professor Schaefer constructed a very simple set consisting of four resonators, and in these he has the range of from 100 double vibrations to 1,000 double vibrations. Dr. Sonnenschein had received a letter from Professor Schaefer two days previously in reply to one of his asking if there was any work that he knew of along the line he and Dr. Minton were carrying out, and Professor Schaefer said he knew of no such work. They admitted that there might be errors. They had checked up the work and would be glad to know if others would do so and tell them their results. It was interesting to test the work out and find out whether it was worth anything from a practical standpoint.

It was very interesting to know the intensity amplification as it appears in a closed room and as it occurs out of doors. In the open the higher fork, namely, small c^3 , seemed to be amplified only five times. If they struck a high pitched fork it could be heard at a distance of five meters indoors, while out of doors one can hear the low tone a great distance.

One thing that was hard to explain was the fact that the lower tone and the higher tone were the least amplified and the middle the most, as shown by Schaefer long ago. The fact that out in the open air the intensity of the amplification is so tremendous, whereas in the room, to the examiner and the patient, the amplification for the low tones is so small, was very interesting.

What Dr. Holinger said about the practicality of the test as devised by Bezold was absolutely correct. Bezold's work was wonderful, and they were not attempting to change any

test, but felt that it would be interesting to find out if the fork could be used in a different way, especially since some European workers had condemned the Rinne test so strongly.

Dr. Sonnenschein agreed with Dr. Shambaugh regarding the value of the Rinne test and approved the simplicity of the tests, but this work was for the purpose of satisfying themselves. If anyone would check them up and disprove their results they would be glad to know the truth.

Dr. Shambaugh had called attention to the fact that it might be possible to test patients with the resonators who apparently did not hear the forks at those pitches. That might not prove whether there were gaps, for it is necessary in some cases to increase the tone intensity 100,000,000 times before the patient can appreciate it. In cases where there was impairment and not total loss of hearing of tone it might be of assistance.

DR. GEORGE E. SHAMBAUGH presented a series of lantern slides illustrating the distribution of blood vessels in the internal ear and discussed the subject of the circulation of the labyrinth.

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XXIV.

DEPRESSED NASAL DEFORMITIES. A COMPARISON OF THE PROSTHETIC VALUES OF PARAFFIN, BONE, CARTILAGE AND CELLULOID, WITH REPORT OF CASES CORRECTED WITH CELLULOID IMPLANTS BY THE AUTHOR'S METHOD.

By JOSEPH D. LEWIS, M. D.,

SURGEON-IN-CHIEF, DEPARTMENT OF EYE, EAR, NOSE AND THROAT, MINNEAPOLIS GENERAL HOSPITAL,

MINNEAPOLIS.

The common types of depressed nasal deformities may be classified as (pronounced) retroussé nose, saddle nose and depressions of the nasal tip. Other varieties, usually resulting from injuries, are seen as combinations of these types.

One of the following three methods may be employed for the correction of external nasal depressions by buried prosthesis:

1. Subcutaneous injections of paraffin.
2. Autogenous transplants of bone or cartilage.
3. Celluloid implants.

These methods will be considered separately, in the order mentioned, and discussed on a basis of comparative value.

PARAFFIN.

The correction of depressed nasal deformities by internal prosthesis had its beginning in 1900, when Gersuny¹ injected subcutaneously white vaselin (also known as medicinal vaselin, which melts at 104° F.), for the correction of a saddle nose. Shortly afterward, Delangre,² for the same purpose and in like manner, used spermaceti. Eckstein,³ in 1901, used pure paraffin with a fusing point of 140° F. Later observers, however, found that a mixture of vaselin and paraffin having a melting point ranging from 110 to 136 degrees F. is preferable.

The ease with which nasal depressions, even of marked degree, may be restored to normal contour by the injection of paraffin gave to this method an immediate and wide vogue, and reports of many cases in which this material was used appeared in the literature of the subject.

The paraffin method, largely because of the simplicity of technic, was promptly adopted by charlatans, who recklessly abused the procedure; but, even in competent hands, this method did not long survive, owing to the appalling end results noted in a considerable percentage of the cases.

Harmon Smith⁴ has published a very comprehensive statistical report, based on information collated through a questionnaire, comprising 1,252 cases (exclusive of his own) in which paraffin prosthesis was used. The following is the statistical summary deduced from answers received from forty-one different observers: At the time of compiling the data 1,000 of the cases reported were entirely successful; in 104 the results were unsatisfactory. Two of the 104 cases resulted in blindness, in 55 sepsis and extrusion of the mass followed, in 22 the paraffin lodged in the wrong location, and in 7 the mass subsequently shifted. In his own 200 cases, in which paraffin was employed, unsatisfactory results occurred in two.

The melting point of the mixture of vaselin and paraffin injected ranged from 103 to 136 degrees F., the majority of the observers using a mixture having a melting point of 110° F., as originally suggested by Smith. Thirty-one of the forty-one observers considered the procedure practicable in the hands of an experienced operator, if proper precautions were

observed, the remaining ten regarding it as dangerous and in most cases unwarrantable.

Connell and Kolle,⁵ adding to Smith's statistics of the untoward sequelæ following the use of paraffin, have tabulated a total of twenty-two complications which may follow its use. Later consequences, not included in the aforementioned reports, have appeared in the form of keloid and paraffinoma.

In my earlier work paraffin, having a melting point of 120° F., was used in six cases for the correction of concave nasal deformities of moderate degree, fortunately, without any untoward results; but the evidence before us seems sufficient to condemn a procedure which all too often has been followed by disastrous consequences.

BONE.

Bone grafts may be obtained from the rib, crest of the tibia, ilium, free vertebral border of the scapula, as suggested by Lothrop,⁶ or from the turbinal bodies.

It is a well recognized fundamental principle of bone surgery that successful bone transplantation is largely dependent on the osteogenetic activity of other bones to maintain continued viability of a graft. Therefore, it is essential to permanence that a graft used to restore nasal contour be placed in close juxtaposition to the frontal bone, because osteogenesis provided by the small and thin nasal bones is inadequate. At best, bone to bone contact can be effected only at one end of the graft. The proximal portion, which rests free in the soft tissues at the nasal tip is, therefore, removed from any contact with the bone.

We shall see, as we proceed, that the opinions of observers are divided respecting the ultimate fate of bone grafts transplanted into the nasal tissues, as well as regards the influence of the periosteum on their permanence.

Carter⁷ reports cases of depressed nasal deformities corrected with bone prosthesis which show no clinical changes after many years. His radiographic studies indicate that the outer layers of bone transplanted into the nasal tissues remain unchanged, whereas the central portions, being more remote from the circulation, reveal definite signs of absorption, which he thinks is probably due to impaired nutrition. At that time (1910) he believed the periosteum essential to the viability of

bone grafts; also that contact with the raw frontal bone is of the highest importance—being secured only when the periosteum has been elevated and the underlying bone denuded with suitable instruments.

As to the value of periosteum, this writer⁸ subsequently revised his views and advocates its removal for the reasons that he believes bare bone is better nourished by the connective tissue which soon surrounds it and, since there is no desire to grow new bone, the osteogenetic layer of the periosteum serves no function. He summarizes additional reasons as follows: "The periosteum is removed from the grafts because: (1) It is easier to resect a rib without injuring the pleura by shelling it out of its periosteum, and besides, it is desirable to leave the periosteum for the reconstruction of the rib. (2) The bare bone is more quickly and surely nourished by the connective tissue envelope which quickly surrounds it. (3) The osteogenetic layer of the periosteum can be dispensed with since we have no desire to grow new bone, but to nourish the transplanted tissues."

Carter⁹ says further: "The influence of the periosteum as an osteogenetic factor is pronounced, and its absence is equally marked. This osteogenetic function is more marked the younger the patient. At the same time I say that I have not found the periosteum necessary in correcting nasal deformities, but the transplants are in a more flourishing condition in those cases where I have preserved the membrane."

Subsequently Carter¹⁰ proposed a modification of the older method, advocating the use of a graft composed of one inch of periosteum covered rib and one-half inch of costal cartilage; the object of this is to supply bone where it normally exists with the cartilaginous portion extending into the nasal tip to preserve its normal flexibility.

For the same reasons, Cohen¹¹ also favors a rib graft composed of conjoint bone and costal cartilage.

More recently Carter¹² states: "My clinical cases appear to show that bone is formed by the so-called periosseous osteogenetic layer of the periosteum, and that the cells of this layer are protected and their growth limited by the connective tissue."

It now seems pertinent to the subject under discussion to

set forth the facts which have led other investigators to the opposite view respecting the ultimate fate of transplanted autogenous grafts and to inquire further into the value of the method.

According to Ferris Smith,¹³ * * * "free plants of bone with or without periosteum into the soft tissues are slowly absorbed. Bone which is turned with a flap without separation of its periosteum, but unattached to other functioning bone, is partly or wholly absorbed, leaving a dense scar which furnishes some support. The difficulty in shaping or handling bone under these conditions practically excludes it as a means of support."

Sheehan¹⁴ also holds that bone transplants, with or without periosteum, are often slowly absorbed, and therefore believes that the method should be discarded.

The exhaustive experimental studies on dogs conducted by Davis¹⁵ to determine the comparative permanence of free bone and cartilage transplants throw considerable light on the relative value of the two. The grafts of cartilage and spongy bone were taken from the rib and the hard bone from the fibula. The plants were inserted beneath the periosteum of the temporal bone, a part of which was placed in contact with the denuded parietal bone, the remaining portion being buried in the temporal muscle. He showed that under the same conditions the behavior of transplanted cartilage and spongy rib bone is quite different; that the cartilage retains its original size, while bone shows a tendency to absorption, which slowly progresses. Later,¹⁶ reiterating these views, he summarizes his conclusions as follows: * * * "my experience has been that a free bone transplant in soft parts will eventually be absorbed and, when in contact with bone at only one end, will eventually atrophy. * * * Those who begin with the use of bone will eventually turn to cartilage as the best material for the construction of a framework for the nose."

The opinion of Gillies,¹⁷ based on a broad experience in the use of bone grafts in various parts of the face and head, is that they eventually absorb and are replaced by a deposit of fibrous tissue, the clinical results depending on the amount of new formed tissue, which, in a fair percentage of the cases, is sufficient to leave the cosmetic result unimpaired to any appreciable degree. Bearing on this point, Hays¹⁸ has described a

case in which three months after a bone graft had been placed in the nasal tissues, it was removed because of persistent infection, and he was happily surprised to find that sufficient connective tissue had been deposited to fill the original concavity.

I also have observed the same phenomenon following the extrusion of a gold implant which had remained in the nasal tissues for several years. Similar observations have been reported by others in the literature on heteroautoplasty.

Thus there is a preponderance of evidence, deduced from the lessons drawn from animal experimentation and clinical observations, which strongly speaks against bone transplantation for the correction of nasal deformities. Seemingly, the method has proved disappointing for manifold reasons, to wit: (1) It is quite impossible to fashion a bone graft so that the result will impart a naturalness to the nose; (2) in order to effect contact with the frontal bone—the source of osteogenetic activity—the plant, in arching across the infraglabellar notch, gives to the nose an appearance similar to the classic type of the ancient Greeks, which does not appeal to the modern esthetic sense; (3) it would seem that depressions of the nasal tip are hardly amenable to correction with bone, for, if viability depends on contact with other bone, necessarily the graft must extend from the tip to the frontal bone, thus elevation of the dorsum of the nose would follow and largely impair or even negative the cosmetic result; (4) infections about the rib area have not been uncommon, and even more serious complications have followed as a result of wounding the pleura, including pneumothorax and empyema. While not of frequent occurrence, the possibility of these complications must be considered; (5) two other factors which may act as deterrents to both patient and surgeon are the inconvenience to the patient occasioned by the operation and the mutilation of the chest wall attending it; (6) and finally, the fate of bone transplanted into the nasal tissues is, at best, very uncertain, the consensus of competent opinion being that it is eventually absorbed.

CARTILAGE.

Cartilage transplantation is a method originally evolved by von Mangoldt¹⁹ in 1899, who used a disk of rib cartilage to

supply tissue insufficiency in an external operation on the larynx. His method, slightly modified, is still the accepted one.

Nélaton²⁰ was the first to employ rib cartilage for the purpose of supplying nasal deficiency. The cartilage was first planted into the forehead, between the skin and the periosteum, and later a pedunculated flap composed of skin, cartilage plant and periosteum was detached and swung down into position, the embedded cartilage serving to prevent sinking of the newformed nose.

Grafts of septal cartilage claim but passing interest inasmuch as there is complete unanimity of opinion among observers that they are always absorbed, fading away so promptly that fibrous tissue has not time to replace the departed visitor. Furthermore, since septal cartilage cannot be obtained sterile, the inseparable element of infection is introduced, rendering the procedure surgically unsound. Cartilage from this source being largely composed of yellow elastic tissue probably satisfactorily explains its inherent absorptibility. On the other hand, cartilage from the rib or sternum may be obtained uncontaminated, and, if desired, with more or less of its perichondrium intact.

The viability of cartilage is not dependent on either contact with bone or cartilage, living, according to Ferris Smith; by lymph absorption—remaining as before introduction when divested of its perichondrium and probably growing when it has been preserved.

As a result of the knowledge gained from a study of a series of twenty-four experiments on dogs, supplemented by his clinical observations, Davis²¹ considers transplantation of rib cartilage a meritorious method for the correction of saddle nose. He has determined by microscopic studies that transplanted cartilage becomes surrounded by a zone of loose connective tissue containing blood vessels, the vascularization varying according to the length of time a plant has been imbedded. The cartilage cells appeared normal, and there were no evidences of absorption, degeneration or increase in the length of the plant—in a word, this competent research worker concludes, from these and other animal experimentations previously referred to, that perichondrium covered cartilage, when transplanted into flaps with skin on two

sides and having an adequate pedicle supply to provide nourishment, is a feasible surgical procedure.

Beckman's²² observations lead him to the conclusion that cartilage is preferable to bone as a supporting material for the relief of depressed deformities of the nose and that it is neither essential to preserve the perichondrium nor to have contact with bone or other cartilage to maintain viability.

As has already been pointed out, Gillies likewise prefers cartilage to bone. As to the permanence of cartilage, he says: "In only a few of the homologous grafts has the cartilage become replaced by fibrous tissue as a late sequel." The longest period of time that a graft had been under his observation is three years.

Cohen²³ states that in his experience the use of cartilage alone has not proved wise for the following reason: "Such grafts never form union with bone and are, therefore, never firmly fixed on the nose; they are always absorbed to a greater or less degree."

Selfridge²⁴ also has noted the absorption of cartilage transplanted into the nasal tissues, particularly instanced in a case reported where three attempts were made before he attained his end. First, a piece of cartilage taken from the rib and sternum was absorbed. Here, as in another case reported in the same article, he met with infection of the rib area. At the second operation he transplanted septal cartilage which became infected and it was necessary to remove it. Finally, again using sternum cartilage, a satisfactory result rewarded his pertinacity.

A survey of the foregoing opinions of those who have studied the comparative merits of bone and cartilage brings us to an apparent point of agreement, to wit: That cartilage is preferable to bone as a supporting material for the correction of depressed nasal deformities. But, as we have seen, there is a divergence of opinion as to the permanence of transplanted cartilage. This important question remains to be definitely determined by the test of time and therefore we must await tomorrow's answer.

In some respects cartilage prosthesis has advantages over bone. In other ways several distinctly weak features are

attached to the method, namely: cartilage lacks sufficient rigidity to provide adequate support for marked nasal depressions because the concavity cannot be spanned, and therefore correction must be accomplished by filling the depressions with cartilage. Although more easily shaped than bone, the fragility of cartilage renders it liable to fracture while being formed or as a result of manipulation incident to introducing it into the nasal tissues.

Cartilage, as we know, is more resistive to infection than bone, but when it occurs, as Gillies and others have pointed out, suppuration and necrosis may follow, and as a consequence a secondary deformity may be the terminal result.

Gillies has demonstrated that the chest wall remains permanently weakened after the removal of thoracic cartilage, because this tissue does not replace itself.

In final analysis one may be led to the conclusion that neither bone nor cartilage—in other words, autogenous tissue grafts—is to be viewed as a wholly satisfactory method for the relief of depressed nasal deformities.

CELLULOID.

The scant recognition accorded the celluloid method in America has, with few exceptions, been expressed in words of disapproval, the idea being rejected on the theory that foreign bodies are not tolerated by the tissues.

The general attitude displayed toward celluloid as a supporting material is reflected in the following quotations from the writings of several contemporary authors:

Ferris Smith:¹³ "A century of experience with all kinds of inorganic substances planted in soft parts has served to prove that only exceptionally is a foreign body kindly borne by the tissues: gangrene, skin atrophy or expulsion of the piece has been the usual experience. Recent workers make different claims for celluloid, and there are still enthusiastic supporters of hydrocarbon preparations, but after all is said both are foreign bodies."

Sheehan:²⁵ "Celluloid plates, * * * cannot be too forcibly condemned. They act as an irritant to the tissue, generally sooner or later giving trouble by abscess formation, gangrene, atrophy of skin and finally expulsion."

Beck:²⁶ "I have had no personal experience with celluloid implants, having rejected them on the ground that they were foreign bodies and would not be assimilated as well as the bone or cartilage graft, nor even as well as the paraffin."

But as these assertions are not based on any actual experience with celluloid or supported by the results of others who have employed this material, they fail to carry conviction. Nor can we view such postulates as in line with the spirit of scientific progress.

In expressing a preference for autogenous grafts, Gillies¹⁷ believes that inorganic substances act as tissue irritants and ultimately the tissues attempt to remove them—in fact, he found that the greater number of his celluloid implants had to be removed within two months of their insertion. While the opinion of one so finely qualified by exceptional experience carries great weight, there are several cogent reasons why failures should attend his endeavors, and therefore his results should not be taken as a true criterion. It is not surprising that he has recorded unsatisfactory results with the use of celluloid, nor is it difficult to see why this should be when we remember that the implants were introduced into tissues which previously had been traumatized by wounds of war, involving both cutaneous and deeper structures, producing, as a result, old scars, hematoma, impaired blood supply and, as a consequence of tissue devitalization, he constantly was troubled by infection. With the exception of one nasal case the operations in which celluloid was used were in other parts of the face.

Notwithstanding the adverse conditions under which he so patiently labored, two of Gillies' celluloid implantations proved successful: one plate healed in after an abscess, which developed over it, was drained.

Seemingly, this investigator, realizing that he labored against almost insurmountable obstacles, has not lost faith in celluloid as a prosthetic material. as, at his request, MacBride of the Imperial Research Laboratories is now carrying out a series of experiments on the implantation of collodion into the ears of mice.

The genesis of the celluloid plate idea is accredited solely to Fraenkel,²⁷ who used plates of this material to fill skull

defects. This fine example of originality and ingenuity has few parallels in the annals of surgery. He found that celluloid plates, when snugly fitted, cause no reaction in the neighboring tissues years after their application. He cites two cases of cortical epilepsy in which the skull defects produced by decompression were replaced with celluloid plates. The results were good seventeen and twenty-five years, respectively, after operation. In referring to Hintersteisser's brilliant results, he says that this observer, who has had wide experience with the method, never found reason to remove a depressed celluloid plate from the skull.

Föderl²⁸ was the first to employ celluloid in plastic operations for the restoration of the nose, using a thin sievelike triangular shaped plate which he interposed between the periosteum of the skull and the skin of the forehead. Allowing the wound to heal, he completes at a second operation by cutting a pedunculated graft containing the skin, periosteum and imbedded celluloid plate, which is brought down and stitched into the desired position to replace the destroyed nose. The thin celluloid plate, being easily bent, serves to produce the desired shape and also to prevent sinking of the bridge of the new-formed nose. He reports two cases in which this method was used. In one, the greater part of the nose had been removed on account of a tumor, the other being a case of destruction of the nose by disease. In both cases the immediate results of the plastic operations were good, but the first patient died of a recurrence of the tumor. In the second, the shape of the nose was not entirely normal, and on account of the marked tissue destruction another plastic operation would be necessary around the mouth. The case was a very unfavorable one, and he thinks the result as good as could reasonably be expected or obtained by any method.

Koschier,²⁹ following Föderl's technic, describes two cases operated on by his method. In one the disfigurement was due to rhinoscleroma, the other being caused by lues. The results were excellent in both cases. On the whole, the author concludes that the celluloid plate method gives better cosmetic results, with a very simple operation, than other plastic methods done for the same purpose.

Thompson³⁰ has shown that celluloid filigree plates used in

operations for large herniæ and plates which are serrated or perforated near the margins, in operations on the skull, revealed no evidence of erosion after being in the tissues as long as three or four years. He found that when buried aseptically and unperforated the reaction caused by celluloid is so slight as to pass unnoticed, nor does it afterward cause any inconvenience.*

In expressing his preference for the celluloid method, he says: "Celluloid will be found advisable in preference to other material, such as transplanted bone phalanges, pieces of rib, etc., where the use of these is indicated in plastic operations on the nose for restoring the bridge, * * * producing a more artistic result, indeed I think a satisfactory one, which is more than can be said for the other crude procedures; moreover, it is not so liable to the necessity for removal for necrosis, with all its undesirable consequences, as in the other case."

Monks³¹ was the first in this country to use celluloid for the restoration of nasal contour—in fact, he was the first to transplant this material directly into the nasal tissues. He has reported two cases of saddle nose successfully corrected with celluloid implants, introduced into a tunnel made by way of an incision at the tip of the nose.

New³² implanted pieces of celluloid into the tissues over the nose in six dogs. In two, the wounds became infected; in the remaining four, the celluloid healed in with only slight reaction. The impressive results of these experiments led him to employ celluloid implants for the correction of depressed nasal deformities in man, in which the results were uniformly satisfactory in his five reported cases. He believes celluloid is preferable to either bone or cartilage.

The prejudice against celluloid has arisen wholly as a result of including this material in the category of foreign bodies which we know are not tolerated by the tissues and therefore have proved consistently disappointing. But it seems to have been demonstrated with certainty by a host of investigators and abundantly confirmed by my own results, that celluloid,

*A perusal of Thompson's illuminating article, dealing with the uses of celluloid in various parts of the body, may prove of interest to the general surgeon.

for reasons as yet not fully determined, is not a tissue irritant and therefore shows no tendency to extrusion, if introduced in reasonable quantities, through avenues amenable to sterilization and when the suture line is well removed from the prosthesis.

In sum, the evidence vindicating the celluloid method appears so ample and conclusive that I firmly believe it is destined to supersede all other procedures.

TECHNIC.

In striving to avoid a more or less conspicuous scar following an incision made for the introduction of a nasal prosthesis operators have shifted from one part of the nose to another.

The oldest method of effecting a subcutaneous tunnel is by way of a transverse incision at the root of the nose corresponding to that point where the bridge of a spectacle rests. The method has lost its former popularity for the following reasons: A disfiguring scar remains if not concealed by a spectacle bridge; the angular branches of the facial arteries are necessarily severed and require buried ligatures—a step not infrequently followed by infection; the angle formed by the glabella and bridge of the nose (infraglabellar notch) calls for a wide dissection over the nasal dorsum in order to gain space to extend the pocket to the tip of the nose. This, in turn, means that there is little to prevent lateral displacement of the prosthesis. The suture line being very near, or directly over the prosthesis, is distinctly disadvantageous, for the reason that the blood supply, having been impaired, aided by the pressure of the underlying prosthesis, the somewhat devitalized tissues fail to unite or the sutures yield from lack of resistance.

Recently Frank and Strauss,³³ aiming to minimize the scar, have proposed a modification of the preceding method, which briefly described is as follows: The initial incision is made in the eyebrow (in operations for broad nose, access is gained through both eyebrows), from which point the tissues over the infraglabellar notch and dorsum of the nose are elevated with specially designed angular elevators and cutting instruments. The sole advantage suggested by this method is that the scar is concealed by the eyebrow. But in entering from this point lies the apparent weakness of the procedure, as it

imposes an extremely difficult, perhaps impossible, technic to follow the contour of a hump or depression of the nose to the nasal tip with angular instruments without mishap. Furthermore, it follows that to introduce a prosthesis into a tunnel thus made would indeed prove a tedious technical step.

Monks³¹ advocates making the primary incision in the under surface of the nasal tip, from which point the tissues over the dorsum of the nose are released to form a pocket. This method has the virtue of simplicity and being free from conspicuous scar, closely approaches the ideal. However, as all internal prostheses exert a strong tendency to move in the direction of least resistance—in this instance, toward the initial incision at the nasal tip—the support afforded at the suture line often is inadequate, as previously pointed out, to prevent extrusion. I have thrice experienced such a misfortune after introducing a celluloid implant by Monks' method, and, I may add in this place, that several others who have had experience with implanted celluloid have emphasized the importance of having the material completely covered with tissue and also have stressed the danger of placing sutures close to the implant.

As a subcutaneous approach to the tissues of the nasal dorsum, Gillies favors making vertical incisions in the vestibule of each nostril near the junction of the skin and mucous membrane which are united by dividing the columna at its attachment to the upper lip. The columna is then dissected free, seized with a suitable instrument and drawn upward to expose the free border of the columella, above which a tunnel is effected toward the nasal arch. This elaborated surgical technic, which is really a double intranasal incision, plus a transverse division of the columna, seems altogether too extensive, therefore the procedure is likely to be appraised as one of doubtful value.

The intranasal method was evolved to avoid a cutaneous scar, but having several distinct disadvantages the measure has met with opposition because: The technic is difficult; it does not insure against lateral displacement of a prosthesis; the material introduced cannot be closed in with sutures; and, *a fortiori*, the method introduces the added and inseparable element of the danger of infection. Therefore it

would seem wise to avoid a method which is not in obedience to the well recognized principles of modern surgical procedure.

I have elsewhere³¹ fully detailed the operative technic of a procedure for the introduction of nasal prostheses which seemingly leaves little to be desired, surgically or cosmetically. Briefly, the technical steps of the operation are as follows: A vertical incision is made in the lower half of the *columna nasi* (A.A. Figs. 1 and 2), the lips of which are undermined laterally toward the nasal vestibules.

Then, by upward cuts with a pair of small curved blunt scissors, the nasal tip is undermined and converted into a hood (B.B. Figs. 1 and 2). With the same scissors, introduced on the flat, the hood is elevated and the tunnel dissection continued as far as desired toward the *infraglabellar notch* (C. Fig. 2).

Summary of advantages: By first packing the anterior nares with gauze or cotton, the field is rendered amenable to sterilization; local anesthesia is wholly adequate; by working through the soft tissues at the base of the nose the parts are easily manipulated. Therefore it is not difficult to follow the contour of the nasal dorsum and, with little care, to carry the tunnel dissection toward the nasal bones without departing from the midline—hence, lateral displacement of a prosthesis is prevented; the support furnished by the tip hood secures against extrusion; the two or three sutures required to close the initial incision are well removed from the tunnel containing the prosthesis; and, lastly, the soft tissues of the *columna* promptly heal without scar (Fig. 3). In short, my results with this method lead me to believe that it is hardly possible to exaggerate its advantages.

REPORT OF CASES.*

Fig. 4 (Case 1).—A. A., aged 23, in 1909 sustained an injury by being thrown against a telegraph pole and again in 1916 during a football game, producing a depressed fracture of the right nasal bone and a dip between the nasal bones and the tip. June 5, 1918, the right nasal bone was refractured and reset by an intranasal operation. Six days later a celluloid implant was introduced through a transverse incision over the bridge of the nose to restore contour between the tip and nasal bones. This case has no special interest

aside from showing the length of time—more than four years—the celluloid implant has remained in situ, and the conspicuous scar produced by the transverse incision over the nasal bridge.

Fig. 5 (Case 2).—F. K., aged 15. Saddle nose of moderate degree. No history of injury being obtainable, this case is classified as a doubtful congenital deformity. Operation, July, 1921, celluloid implant introduced by author's method. Note absence of scar and naturalness of the nasal contour.

Fig. 6 (Case 3).—O. B., aged 60. Saddle nose of moderate degree resulting from a septal abscess. Procedure same as in preceding case.

Fig. 7 (Case 4).—H. G. D., aged 33. Depression of the nasal dorsum following a submucous resection of the nasal septum in 1915. Celluloid implant introduced as in preceding cases. Note the change in the shape of the nostril.

Fig. 8 (Case 5).—P. H., aged 27. Saddle nose and flattening of nasal tip due to injury in 1919. Celluloid implant shaped to correct the dorsal depression and elevate the tip.

Fig. 9 (Case 6).—B. D., aged 16. Injury to nose resulting from a fall, producing a retroussé deformity. A septal deviation almost entirely obstructed the left nasal chamber. Submucous resection of the nasal septum; later a celluloid implant was introduced by the author's method.

Fig. 10 (Case 7).—B. N. S., aged 9. Congenital depression of the nasal tip. April, 1920, celluloid implant introduced through a bridge incision (note scar) and removed a few days later, as it became displaced toward the suture line and was exposed. April, 1921, celluloid implant introduced into the tip by author's method, which was not sufficiently large to correct the deformity. February, 1922, attempt was made to remove implant introduced ten months previously, preliminary to inserting a larger one, but, owing to its having become securely encapsulated this was not possible, so a second implant was superimposed upon the first. (The two implants are shown in accompanying skiagraph.)

Fig. 11 (Case 8).—A. K., aged 26. Congenital hump and depression of the nasal tip. Through a columnar incision

*Only those of my twenty-three cases of celluloid implantation which have some special interest are herewith reported.

(A. Fig. 1) the hump was reduced with special rasps and immediately afterward the tip elevated by a suitably shaped celluloid implant.

Fig. 12 (Case 9).—J. C., aged 20. Congenital depression of the nasal tip. July, 1921, celluloid implant introduced by author's method, which was removed February, 1922, and another of different shape and somewhat larger introduced. At the time of removal of the first implant it was noted, by the resistance offered, that encapsulation was quite well established. Examination of the first implant under magnification showed no erosion.

Fig. 13 (Case 10).—K. A. M., aged 38. A submucous resection of the nasal septum was done December, 1917. Extensive sloughing followed, which completely destroyed the septum, producing a very ugly external deformity. Lues being suspected, a Wassermann test was made, which proved positive + + + +. Patient consulted me November, 1921, stating that he had received antiluetic treatment. Wassermann negative. November, 1921, a celluloid implant was introduced by the author's method. The result not being entirely satisfactory, the first implant was removed after twenty-five days and a larger one inserted.

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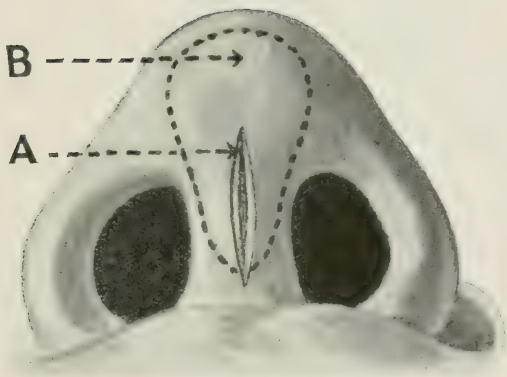


Fig. 1.

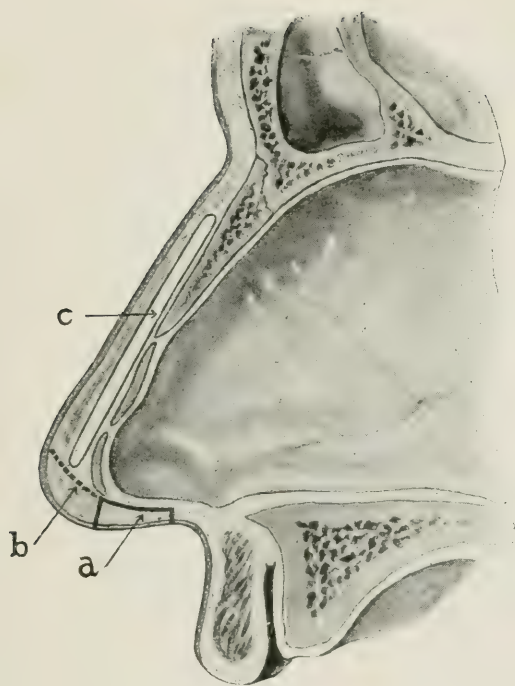


Fig. 2.

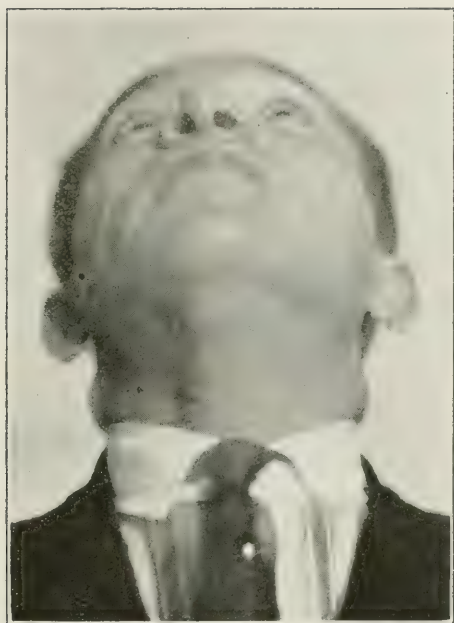


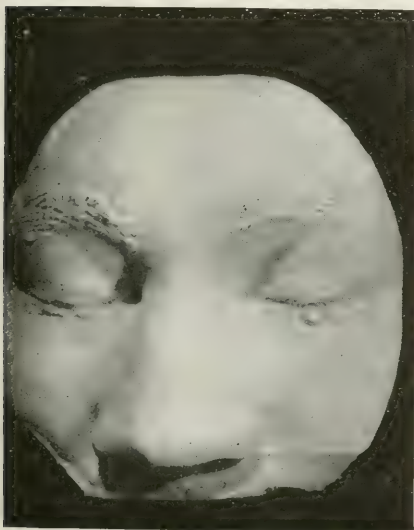
Fig. 3.

Base of nose, showing absence of scar following correction of saddle nose and tip depression with a celluloid implant by author's method. (See Fig. 8, Case 5.)

Fig. 4 (Case 1).



Preoperative Appearance.



Postoperative Appearance.

Fig. 5 (Case 2).



Preoperative Appearance.

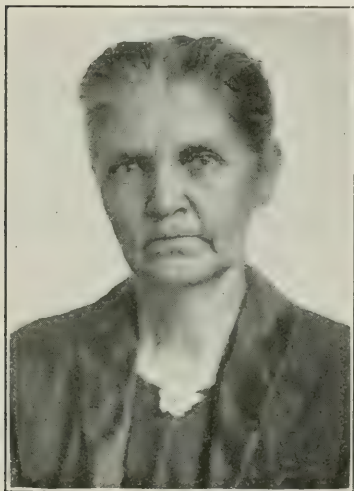


Postoperative Appearance.

Fig. 6 (Case 3).



Preoperative Appearance.



Postoperative Appearance.

Fig. 7 (Case 4).



Preoperative Appearance.

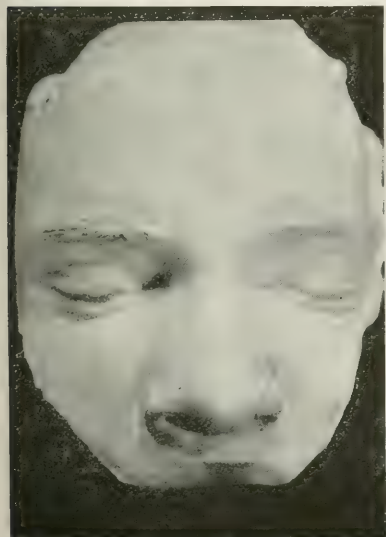


Postoperative Appearance.

Fig. 8 (Case 5).



Preoperative Appearance.



Postoperative Result.

Fig. 9 (Case 6).



Preoperative Appearance.



Postoperative Result.

Fig. 10 (Case 7).



Preoperative Appearance.



Postoperative Result.



Skiagraph showing the two celluloid implants used in Case 7.

Fig. 11 (Case 8).



Preoperative Appearance.



Postoperative Result.

Fig. 12 (Case 9).

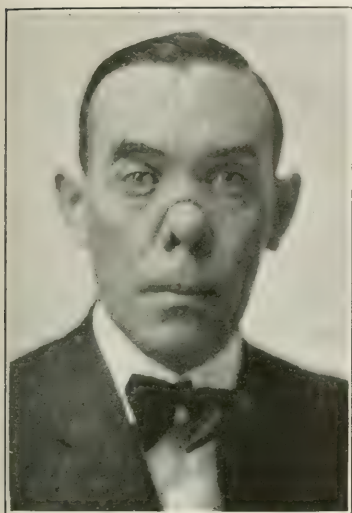


Preoperative Appearance.



Postoperative Result.

Fig. 13 (Case 10).



Preoperative Appearance.



Postoperative Result.

XXV.

THE RELATION BETWEEN THYROTOXICOSIS AND TONSILLAR INFECTION.

BY LOUIS E. BROWN, M. D.,

AKRON, OHIO.

Considering the amount of attention which has been directed toward infection originating in the tonsil, and also the extended study of disturbance of the thyroid function, it seems rather surprising that so little consideration has even been given to the possibility of relationship between the two. It would seem almost inevitable that whichever avenue of approach an investigator used, whether his interest were in the tonsillar condition or solely directed toward a consideration of thyroid pathology, he would sooner or later arrive at a point where the two lines of investigation would intersect. Yet a careful search of the literature reveals comparatively few instances of any such occurrence.

The writer's interest was first aroused to the possibility of some interrelationship between diseased tonsils and disordered thyroid in the routine course of otolaryngeal practice. The extensively reported experiments of Kimball and Marine upon the public school girls of Akron and other Ohio cities have recently called the attention of the medical profession all over the country to the fact that goiter is especially prevalent in this particular section of the United States. In common with the other medical men located here, the laryngologist sees many patients who present some degree of thyroid disturbance, the vast majority of these patients being adolescent girls or young women not many years past puberty. As the writer's interest was primarily in the indications of the tonsillar situation, and the goiter was considered only in the general survey of the patient's health and physical condition, it was some time before the conclusion was thrust upon him that there could be any definite connection between the two.

This is unfortunate in view of the present inquiry, for much valuable and interesting data were allowed to go unrecognized

and consequently unrecorded, which, if available at present, might be of great assistance in illuminating a rather obscure subject.

It was not until many patients—in the course of investigation of their general condition following tonsil enucleation—had reported decided improvement in the goiter itself, and an undoubted amelioration of its characteristic symptoms, that it became evident that these circumstances must be something more than casual coincidence. Nervousness had been greatly lessened and irritability was much less evident, pulse rate was markedly lower, and there had been a gratifying increase in weight. The thyroid enlargement in many cases had undoubtedly decreased, and the whole physical and mental condition was much benefited.

Confirmation of these observations came from another source—that relatively small class of patients who presented themselves for tonsil enucleation giving a history of previous thyroidectomy without any marked amelioration of the goiter symptoms. Relieved of their infected tonsils these patients reported a complete clearing up of all the thyrotoxic symptoms which had persisted after the thyroidectomy.

The repeated occurrence of these cases so impressed the writer with their significance that he resolved to extend his researches outside his own practice, and find out—if possible—whether other men working along the same general lines had had opportunities for making observations of similar character. With this purpose in view, he prepared a brief questionnaire covering those points on which he most desired additional information, and sent it to twenty-eight practitioners whom he believed to be so situated as to be able to supply the information he was seeking. Surgeons, internists, and ear, nose and throat men were represented in the list, the idea being to get as broad a range of opinion and as many different points of view as possible.

The response to the appeal was very gratifying. All the letters were answered, and although the majority modestly disclaimed possessing any information which they deemed valuable, the recipient of their kindness is compelled to differ with them. At the time the questionnaire was sent out the writer had found very little in the literature which had any bearing

upon his hypothesis, and although further search revealed a number of records and reports along these lines, the personal communications still remain of the highest value and interest.

Of the twenty-eight men to whom these inquiries were sent there were only six who felt unable to give any definite reply to the questions. Several of the remaining twenty-two were not in a position to give exact answers to the questions as arranged in order, but the general observations and conclusions they did give were very valuable and helpful. The results of the questionnaire may be summed up, using the different questions as headings:

1. How many cases of goiter have you had in your practice, in which, according to the patient's history, the symptoms accompanying the goiter were apparently temporarily increased during an attack of tonsillitis?

Thirteen replied positively that symptoms were always increased during an attack of tonsillitis, and but two had failed to observe any relationship between the two conditions. David Marine has often observed the phenomenon, but does not believe there is anything "specific in the reaction of these (goiter) cases to tonsil infection." Plummer of the Mayo Clinic is of the opinion that exophthalmic goiter "is sometimes aggravated by tonsillitis, while in other instances it is at least temporarily improved," this answer, however, having little if anything to do with the etiologic relationship of tonsillitis and exophthalmic goiter. Allen Graham calls attention to the fact that while tonsillitis undoubtedly aggravates both simple and exophthalmic goiter, these same phenomena have been observed in many other acute infections, "most notably tuberculosis."

2. In how many cases, where adults presented an early stage of goiter coincident with infected tonsils, was the growth of the goiter apparently arrested by the removal of the tonsils?

All the replies to this question might be classed as "affirmative." Comparatively few were able to state an exact number of cases, but the consensus of opinion was undoubtedly that the removal of infected tonsils has a beneficial effect on all types of goiter. Dr. Crile states that "in general, 50 per cent are apparently arrested by the removal of any infection, whether it be teeth, tonsils, sinuses, etc." Allen Graham says that

"it is unquestionably true that some patients who have both goiter and tonsillar infection are improved after tonsillectomy. This is strongly maintained by Dr. H. G. Sloan of this city (Cleveland, Ohio), who nevertheless recognized the importance of eradicating infection in the sinuses or any other focus as well as the tonsils."

Albert J. Ochsner has seen many goiters arrested after removal of the tonsils, but in all of these cases the patients were directed to follow a highly specialized diet and were especially cautioned to drink only boiled or distilled water. Dr. Ochsner would therefore appear to consider the dietary regime as more likely to have effected the improvement. David Marine thought that while few, if any, goiters were actually arrested, many were no doubt improved by the removal of any focus of infection, while O. P. Kimball had "seen two cases of well advanced exophthalmic goiter cleared up entirely on the removal of very bad tonsils, plus the simple treatment of saturating the thyroid gland with iodine." In this connection it is interesting to recall the statement of Marine and Kimball published last year,¹ that of more than 1,000 schoolgirls who took iodine prophylaxis for a period of three years, only five showed an increase in the size of the goiter at the end of the period, and four of these five had enlarged and infected tonsils.

3. How many cases, after a thyroidectomy, in which the symptoms persisted, were relieved by a tonsillectomy?

A wide divergence of opinion was revealed by the answers to this question. Mithoefer of Cincinnati had never observed any such cases, while T. E. Carmody (whose figures are only approximate) had seen two: J. M. Ingersoll of Cleveland estimated the number at about 10 per cent of thyroidectomies, and James J. King of New York had an "impression" that one or two who were not relieved by thyroidectomy were relieved by a subsequent tonsillectomy. While Crile was unable to give exact figures, he attributes the persistence of goiter symptoms after thyroidectomy to one of two factors: "1. Not sufficient thyroid removed. 2. Focus of infection has been overlooked." This is interpreted by the writer to mean that if the focus of infection were in the tonsils the goiter symptoms would continue so long as the tonsil remained unextirpated. J. A. Stucky of Lexington, Ky., on July 9, 1921, had

seen seven cases still under observation where the usual symptoms persisted after thyroidectomy, but "were relieved promptly by tonsillectomy." It is likely that Dr. Stucky may later be able to throw more light on this particular question. Several others had no data of any sort on which to base a reply to this question.

4. How many cases of young girls at puberty, who have a beginning simple goiter, had their tonsils removed in early childhood, or at least before the symptoms of goiter were evident?

The most of those questioned "fell down" on this section. "No data" was the reply of the majority. Dr. C. W. M. Poynter of the University of Nebraska considered it "particularly significant." He believes that "as long as we do not know what makes the tonsil susceptible to infection we should begin by assuming that 'lowered resistance' due to goiter acts, not in a specific way, but as any other devitalizing influence, as bad hygiene, etc." Wallace Irving Terry of San Francisco thinks "it will be interesting to learn in a few years whether goiter with exophthalmus is less prevalent in young women who have had their tonsils removed in early life."

5. In those who have not had the tonsils removed, how many were benefited or cured by a tonsillectomy?

All who answered this question seemed to hold the opinion that in adolescent girls who were developing goiter the removal of infected tonsils invariably had a beneficial effect on the thyroid condition. Dr. Crile says, "We feel that only very frank exophthalmic goiter patients are not cured by removal of focus of infection unless they also have thyroidectomy performed."

6. Have you ever had a goiter case that also had infected tonsils, where, after a thyroidectomy had been done, the patient had no further tonsillar trouble?

The only affirmative answer to this question was that of H. S. Plummer of the Mayo Clinic. All the rest answered no, without any qualifications. The unanimity was decidedly striking.

7. What per cent of goiters, in your opinion, are of toxic origin?

This question was evidently somewhat vague, and consequently those who attempted to answer it found considerable difficulty in making their replies satisfactory to themselves. Charles H. Major judged that about 40 per cent of the cases coming to his clinic are toxic. Allen Graham objected to the term "toxic goiter" as indefinite and "subject to various interpretations." In common with Ochsner, he feels that "the region from which the patient comes" is the most potent factor in the nature of the thyroid disturbance. The other replies estimated the number of toxic cases all the way from 50 per cent to "practically all." Marine thinks "relatively few exophthalmic goiters are of bacterial toxic origin."

Many of those questioned made valuable contributions to the discussion, which were not directly included under the questionnaire. There is evidently a strong feeling that treatment, especially the iodine therapy so successfully administered by Marine and Kimball, or the special diet and regimen as advocated by Ochsner, have a much greater part in effecting cure or amelioration than the removal of infection, in the tonsils or any other location. Terry believes that "focal infections play a considerable part in activating a hyperplastic thyroid, but unfortunately removal of foci of infection is not followed by many cured of the goiter." Marine regards "most of the (goiter) cases as types of exhaustion as, for example, those following fright and prolonged worry," all acting "on the same mechanism within the body." He believes "the suprarenal gland plays a very important role in this syndrome, and it has long been known that this gland likewise plays an important role in infections." He adds that "one must bear in mind that the thyroid hyperplasia in goiter is accompanied by lymphoid hyperplasia irrespective of clinical associations," and this causes him to "seriously doubt that any cause and effect relation between lymphoid hyperplasia and thyroid states will be established."

Dr. Poynter sees nothing in the juxtaposition of the tonsils and thyroid to warrant us in assuming a connection between them, either through lymph or blood channels. He thinks that if any relation exists it will be found to be through internal secretion, but adds that "a very large percentage of hyper-

trophied tonsils in conjunction with enlarged thyroid might be significant."

Emil Mayer wrote that, while he was unable to give any exact figures, his experience led him to believe that "an early operation on the tonsils would have a very beneficial effect upon the (goiter) patient, retarding the growth, or even entirely preventing the further advance of a goiter that often is of toxic origin."

A careful examination of the replies as a whole would seem to lead one to the conclusion that most of these men have given little consideration to the possibility of an interrelation between infected tonsils and thyrotoxicosis. The consensus of opinion may perhaps be pretty fairly summed up in the words of Allen Graham, who wrote:

"Regarding exophthalmic goiter, in the vast majority of cases, neither on history nor physical examination can the tonsils be incriminated as the chief etiologic agent, nor the factor that prolongs the syndrome, even granting that it (tonsillitis) might aggravate the symptoms when it occurs. On the other hand, a considerable number of patients date the onset of their trouble from a systemic infection, not necessarily of tonsillar origin. Also a large number of cases develop in which the infection plays little or no part, so far as can be determined by history and examination."

The function of the normal tonsil is not fully known, indeed there is relatively firm ground under the feet of those who maintain that there is no such thing as a normal tonsil, inasmuch as practically each one removed can be proved to be more or less pathologic. Anyone who has investigated the anatomy of the tonsils will immediately perceive how liable they must be to infection with all kinds of bacteria. The tonsils occupy an "open" position in the throat—that is, no food or air can find its way to the stomach or lungs without first passing these tissues. Moreover, deglutition opens up the tonsillar fossæ in such a way that the mouth secretions continually come in contact with them. We know that the tonsil encloses a number of deep crypts lined with highly sensitive epithelium. A cross section of the tonsil mass will disclose how these fossæ with their covering of columnar epithelium reach far down, sometimes to the very root of the tonsil itself.

Not infrequently one crypt communicates with others, and their delicate lining epithelium is directly in contact with the tonsil parenchyma, the chief constituent of which is mononuclear cells. Thus a great extent of absorbent surface is always open to infection, and in addition to this the opening of a fossa is apt to become clogged during the progress of any inflammatory process, so that the infectious material which the crypt contains is sealed up within it. When we consider that the tonsil does not secrete and has no avenue of elimination except through the mouth of the fossæ, we can readily comprehend how this infective material must of necessity be gradually absorbed into the system.² Many years ago George B. Wood³ demonstrated that bacteria will generate toxin in the crypts and that if this bacteria is virulent its first point of attack will be the cryptal epithelium. In the large majority of cases they gain access to the tonsillar parenchyma only after their toxins have destroyed the epithelium. When the bacteria have gained access to the tonsillar tissue, they find permanent lodgment only in the germinating follicles. The current in the interfollicular tissue tends to carry the bacteria toward the efferent lymphatics. Thus a definite lymphatic connection may be established with many other parts of the organism.

During the war period the energies of the medical profession were so completely absorbed by the pressing problems of the battle area and evacuation hospital that all mere academic inquiries were laid aside and forgotten. To find anything bearing on a possible relation between thyroid and tonsillar infection it is necessary to search the literature which appeared before the world convulsion of 1914.

Up to the end of the nineteenth century the thyroid had received little attention as a possible factor in systemic disease. Its connection with simple and exophthalmic goiter, with myxedema and cretinism, was fairly well understood, but there had been comparatively few studies made of these conditions outside the regions where goiter was endemic. During the closing years of the century Roger and Garnier⁴ published in *La Presse Medicale* and the *Transactions of the Societe de Biologie* a series of papers on the thyroid gland, one of which dealt especially with the reactions of the gland to general infections, and the pathologic changes taking place at such a

time. Thirty-three postmortem examinations were made, the material being taken from patients dying of such diseases as diphtheria, scarlet fever, cerebrospinal meningitis, smallpox and purulent staphylococcic peritonitis. These authors reported that the condition of the thyroid was about the same under all conditions; that neither the specific character of the infection nor its virulence and duration seemed to make much difference in the condition of the gland under examination. Most of the lesions were characteristic throughout. The glandular hypersecretion appeared less abundant in the diphtheria cases than in those of scarlet fever, but the diphtheria thyroids contained a greater number of desquamated cells and the colloid material was more frequently altered. In one diphtheria case a parenchymatous hemorrhagic area was found in the thyroid.

These authors performed a number of experiments upon rabbits and guinea pigs, finding it an easy matter to infect the thyroids of these animals by injecting a culture into the center of the carotid artery, which had been previously ligated below the junction of the thyroid artery. The infection thus set up was practically identical with that observed in human subjects, although much more acute, and so brief in duration as to give little or no time for organic reactions to be established.

The conclusions drawn by these authors were that during the course of acute infections disturbances of secretion more or less profound occurred in the thyroid, "as in the other glands of the economy." After a period of hyperactivity, a period of diminution will follow or an alteration of function. Just as the liver at such times will secrete abnormal pigments, the thyroid will form colloidal material, atypical and peculiar to these conditions.

Roger and Garnier's investigations apparently attracted little attention in this country, and it was not until Billings, Rosenow and their coworkers began to publish the results of their researches on focal infection that the possible relation of the thyroid to systemic disturbances was brought to general notice in the United States. In his address on focal infection delivered before the American Medical Association in 1914 Frank Billings⁵ reported three cases of rheumatism attended by acute tonsillitis and thyroiditis, attributing all three mani-

festations to a single focal cause. He went on to say that the interest of his clinic was aroused to the possibility of focal infection as a cause of goiter. He reported seven additional cases which "seemed to show that there is an infectious type of goiter with and without symptoms of exophthalmic goiter, which seems to be of toxic origin. The rapid subsidence of the goiter and the symptoms after the removal of the foci of infection in the jaws and tonsils was a surprise." All of these patients gave a history of chronic tonsillitis. Dr. Billings ended by saying that to the list of acute conditions already known to be due chiefly to focal infection we were now justified in adding several others, including "certain infectious types of thyroiditis, with or without hyperthyroidism."

A year before Dr. Billings' address was made Clement F. Theisen⁶ of Albany, New York, published some causes of acute tonsillitis complicated by an acute thyroiditis. These cases offer perhaps the best support which the present writer has been able to find in the literature of a close interrelation between infections of the tonsil and the thyroid. Theisen believed his cases to be "of particular interest from an etiologic standpoint, as in all except one case the inflammation of the thyroid gland occurred with or directly after attacks of tonsillitis. Two of these patients have each had two distinct attacks of acute thyroiditis, each time with an acute tonsillitis, and both have since developed well marked diffuse goiters." While Theisen does not wish to be understood as emphasizing the foregoing facts as important etiologic factors in the development of goiter, he feels that it is by no means impossible that the repeated inflammatory attacks to which the gland was subjected may have, partly at least, been responsible for the subsequent chronic hypertrophy of the thyroid gland.

All of Theisen's patients were young women. Briefly summarized, the cases were as follows:

Case 1: Age 20. Sore throat for several days before examination revealed a typical follicular tonsillitis. Thyroid enlarged and tender on palpation, the hypertrophy increasing during the course of the tonsillitis. The swelling of the thyroid had begun during the third day of "sore throat," and the patient stated that the gland was not enlarged before the attack of tonsillitis. Under the usual treatment for tonsillitis,

with an ice coil about the neck, the attack subsided in about a week. Tonsillectomy was refused, and the next winter the patient again presented herself with a similar attack, "running the same course and again developing with an acute tonsillitis. This patient came to the clinic at regular intervals during the next two years, and while there were no further attacks of acute thyroiditis, she developed a gradually increasing diffuse goiter. It is at least possible that etiologically there is a connection between her attacks of thyroiditis and the subsequent hypertrophy of the gland. There is no doubt that the infection of the gland was each time caused by the acute tonsillitis."

Case 2: Age 22. Same history as preceding case. Patient was practically well in ten days and was not seen again for about two months, when she came to the clinic with typical symptoms of hyperthyroidism.

Cases 3 and 4: 21 and 24 years old. Both stated that before the present attack they had no enlargement of the thyroid. One patient was just getting over a severe attack of acute tonsillitis, and the other was still having an acute attack (no mention of sequelae of hyperthyroidism in these cases).

Case 6: Age 19. Very severe acute tonsillitis, which was followed by the development of acute thyroiditis. The attack ran the usual course and a year later another acute thyroiditis came on with an acute follicular tonsillitis. This patient has been under observation continuously and has developed a well-marked diffuse goiter, which started about six months after her last attack of thyroiditis.

Case 7: Age 30. A very severe acute thyroiditis came on directly after an acute tonsillitis. "There was a good deal of dyspnea and dysphagia in this case, and within a few months after the attack she developed a typical condition of hyperthyroidism."

In concluding this report Theisen remarks that he found very few cases recorded in the literature where acute thyroiditis occurred in conjunction with tonsillitis. A study of his case histories naturally suggests the possibility of overlooking the occurrence of thyroid involvement in acute tonsillitis. It seems reasonable to suppose that recurring attacks of tonsillitis might involve the thyroid and eventually set up a chronic con-

dition of hyperthyroidism, even if the thyroid symptoms were never sufficiently acute to be differentiated during the tonsillitis attacks.

Seven years ago Shurley⁷ stated that his attention was called to the relation of the tonsils to thyroid disease "by the beneficial results of a series of tonsillectomies for the relief of recurrent tonsillitis and quinsy, attended by incipient typical and atypical Graves' disease. The prompt, permanent and prophylactic value of enucleation in this class of cases adds another definite indication to surgical procedure which is given no attention in the literature. As acute and chronic tonsillitis and peritonsillar abscess are recognized as important etiologic factors in incipient exophthalmic goiter, tonsillectomy may then be classified as a prophylactic measure in our new and fashionable department of preventive laryngology."

The discussion which followed Dr. Shurley's paper brought out some interesting data on the relation of tonsil infection to thyroid disturbance. Greenfield Sluder of St. Louis stated that while the clinical relationship between the lymphoid ring and the thyroid gland was not clear in the minds of anatomists, it is an established fact that the lingual tonsil develops from the same branchial arch, and in early fetal life there exists the thyroglossal duct, which is closed early. It is sometimes found in the dissecting room, but the speaker has never been able to observe it in a living subject. He had observed marked improvement in goiters where all treatment had been directed toward the lingual tonsil.

Dr. George B. Wood of Philadelphia reported the case of a trained nurse who had recurring tonsillitis, followed by exophthalmic goiter and hyperthyroidism. After removal of the tonsils the attacks stopped for six months, the goiter began to go down and the exophthalmos to disappear. Then followed a slight sore throat with a less severe attack of hyperthyroidism, and examination now revealed that the faucial tonsils had not been entirely removed, a small piece still persisting in the upper part of the tonsillar recess. When this was removed all symptoms of hyperthyroidism permanently disappeared.

Dr. Woods' explanation of this case was not by a direct relationship between the lymphoid ring and the thyroid gland,

but rather by the presence of an infective process originating in the ring which upset the metabolism of the body so as to produce goiter and hyperthyroidism.

Shambaugh of Chicago was of the opinion that the phenomena of thyroid disease suggested very strongly a condition caused by some focus of infection, as around the teeth, or latent in the faucial tonsils. This latter condition is often overlooked. When the systemic condition develops in connection with a severe attack of acute tonsillitis, the tonsils are often suspected of being the focus, but if the infection is latent and the patient does not complain of throat symptoms, the examiner may not think of the tonsils at all. Chronic tonsillar abscesses are frequently discovered in patients who give no history of "sore throat," and such abscesses are often unrecognized before the tonsils are extirpated.

Evans and his collaborators^s made extensive observations on students entering the University of Wisconsin during the five years 1910 to 1914, inclusive. Wisconsin, being situated in the so-called "goiter belt," offers an excellent field for research on the subject here discussed. The tabulated results of these researches "afforded decided evidence of the actuality of a connection between nasal and throat affections and the large occurrence of thyroid involvement." The recognition of the tonsils as a site of parasitism by *endameba gingivalis* (Gros) in the laboratories of the Universities of Pennsylvania and Wisconsin raised the question of a possible connection of this organism with the thyroid enlargements "because of the evidence obtained of its part in the etiology of other conditions, for example, pyorrhea."

The authors emphasize the fact that in suggesting endamebiasis of the upper respiratory tract as possible causes of thyroid hyperplasia they are not advancing these protozoa as the specific producers of the toxins effective in producing the hyperplasia. If this were true, there is no reason why everyone who is the host of these parasites (a high proportion of all adults) should not also be the subject of thyroid enlargement. Their belief is that the essential toxic factors are really the products of the bacteria associated with the amebæ, and that these bacteria are the variants and the amebæ the con-

stants in many different infections of the mouth, tonsils and so forth.

Acting upon this hypothesis they selected forty-one cases of cryptic tonsillitis with thyroid involvement for special study and treatment by emetin hydrochlorid. Their conclusions touching the relations between tonsillar infection and goiter were that "inability to demonstrate endamebæ in the thyroid gland renders improbable any direct causal relation of the amebic infestation of the tonsils per se upon the development of thyroid disturbances. However, the improvement, morphologically and symptomatically, in the treated cases leaves little doubt as to an indirect relationship. A symbiosis of endamebæ with appropriate bacteria, leading to the elaboration and absorption into the thyroid of selective thyrotoxic poisons, is at least conceivable in explanation of such relation."

One of the strongest supporters of the theory of infective interrelation between the tonsils and thyroid is S. P. Beebe⁹ of New York, who has from time to time put himself on record concerning it. He calls attention to numerous observations on the relation of thyroid disease to previous infections. In this connection it is well to remember that thyroid disturbances occur most frequently in persons of a thymolympathic constitution, and it is these individuals who are most susceptible to infections. The terminal event in hyperthyroid patients is not infrequently an infection which has begun in the tonsil. A large percentage of patients with exophthalmic goiter have enlarged tonsils and adenoids. It is not uncommon to date the beginning of a thyroid enlargement from a particularly severe attack of tonsillar infection.

Infections in the nose and throat are undoubtedly the most common to which the human family is subject, and the tonsil is one of the most important points of entry we have for infections, but in goiter the resultant condition is a hyperactivity of a gland of internal secretion and not a continued infection. It is obviously more difficult to explain such a result than to trace the connection between an acute tonsillitis and a septicemia or an infected joint.

Infection does not in a large percentage of cases produce such an enlargement of the thyroid gland that it would be

recognized as a goiter, and it may be that the thyroid does not react in this manner except in those who are not quite normal in respect to the balance of their glands of normal secretion. If the thyroid secretion is an important element in the defense against infections, it is not impossible that it is stimulated to overactivity when occasion demands, and if the stimulus be often repeated it may lead to changes which we recognize as pathologic. Through the repeated stimulus to overactivity, the gland has become hypertrophied, and its heightened function continues long beyond the stimulus which originally called it forth.

Clinically there is an important relation between the infections in the nose and throat and hyperthyroidism. In patients between the ages of sixteen and twenty-four, from 35 to 40 per cent give a history of repeated attacks of acute tonsillitis and many have enlarged tonsils and adenoids. Rapid enlargement of the thyroid, with characteristic symptoms of overactivity, has often followed immediately after a particularly severe tonsillar infection. Such patients bear these infections badly. Their convalescence is slow, and each attack is accompanied by severe prostrations quite out of proportion to the apparent severity of the infection. Dr. Beebe has observed that the leukocytosis in these cases is lower than that of non-goiterous patients, and that hypertrophied patients often show a marked leukopenia with a relative lymphocytosis indicating some influence on the blood picture operative when the organism is subjected to infection.

The tonsil infections to which exophthalmic patients are so often subject constitute most dangerous and distressing complications and the alert surgeon should always be on his guard against them. If there is an active thyroid intoxication it is seldom wise to enucleate tonsils and adenoids, because such patients react badly to operations of any sort. The anoci-association methods so successfully employed by Crile in thyroidectomy, which Beebe described as "stealing the thyroid," ought always to be employed in all operative measures on cases of this type.

The writer has felt justified in quoting thus at length from Dr. Beebe's excellent paper, as the position he occupies is in

most respects exactly analogous to the writer's own, and the opinions brought out in the discussion which followed the presentation of the paper before the Laryngeal Section of the American Medical Association so closely resemble those expressed by the recipients of the present writer's questionnaire. John F. Barnhill was emphatically of the opinion that the removal of diseased tonsils had no effect upon the progress of the goiter or exerted any beneficial influence upon thyrotoxic symptoms. This was in 1914. In 1920, Dr. Barnhill made an address to this same section on "Surgery of the Thyroid,"¹⁰ in the course of which he said: "In the last six years I have made accurate notes as to the presence of diseased tonsils in all goiter cases. More than 90 per cent of all cases I have examined have had clearly evident disease of the tonsils, and judging from the most modern point of view as to what constitutes a diseased tonsil I think all may rightly have been classed as having foci of infection in the tonsil. In more than 50 per cent of my cases of goiter in which operation was performed during this period, the tonsils were removed before the thyroidectomy, sometimes as long as a year previously, in the hope that the goiter operation might thus be avoided. It seems certainly true that after the thyroid is once diseased the removal of the tonsils has little appreciable benefit on the thyroid disease. Indeed I have seen the thyroid rapidly enlarge and the thyrotoxic symptoms increase after the performance of a most complete tonsillectomy. These observations do not, however, form a good argument against the possibility that the diseased tonsil may have been the original focus from which the thyroid received its infection. Indeed, the frequent presence of infected tonsils in thyroid cases points almost certainly to a connection between the two diseases."

Approaching the question from the thyroid side, we have the assurance of Joseph C. Beck, of Chicago,¹¹ that he has frequently found the removal of one or both tonsils to be far more efficacious in preparing a patient for a thyroidectomy than ligation of the superior thyroid arteries, which is "considered plausible procedure to cure a thyrotoxic disease or to arrest it so as to enable the surgeon to perform a more radical procedure, namely, thyroidectomy." He also found "a fairly good number of thyroidectomized patients in whom the toxic con-

dition recurred, to be much improved or completely cured by tonsillectomy." He believes that in all thyrotoxic cases it is better to proceed with a preliminary tonsillectomy, followed by the proper thyroidectomy than to reverse procedures.

Another point of interest in connection with the thyroid and the laryngologist is the early diagnosis of a thyrotoxic condition. The laryngologist is frequently the first to be consulted in reference to the headache and nervous phenomena, to differentiate between sinus disease, ocular conditions and chronic focal infection, particularly from tonsils.

Dr. Beck supported his contention by reporting the following case:

Mrs. W., age 32 years, has had a somewhat small goiter for several years. As a child had many sore throats, then none for several years. Last winter had two severe attacks of tonsillitis, following which the thyroid gland appeared to get somewhat larger. There appeared also all the classic symptoms of a thyrotoxic state without much exophthalmos. A competent internist counseled very strongly against a major operation at this time. Under local anesthesia he removed her tonsils without any difficulty, either local or general. The thyroid gland receded in two weeks, and her general condition improved very rapidly after that. It is now three years and she has not had any recurrence of her thyrotoxic symptoms. Dr. Beck has had "a fair number of such cases with similar results."

Somewhat similar is a case recently reported by Greenberg,¹² where a small goiter had been present for many years without other symptoms of hyperthyroidism. Following several attacks of "quinsy," typical thyrotoxic symptoms appeared, but were relieved by operation.

The purpose of this article, and of the investigation upon which it is based, is not so much to demonstrate a theorem, of the truth of which the writer is absolutely convinced, as it is to bring together the varying opinions and clinical observations of practitioners who have had occasion to deal with the subject from different angles, and in this way to

stimulate further study, research and discussion of what appears to him to be a highly interesting and significant situation. Beebe stated that his deductions were drawn from the history of approximately 3,500 patients who had thyrotoxicosis, and while it is quite possible for a practitioner to have a number of patients with thyroid disease who do not present infected tonsils or other apparent focal infection, he, nevertheless, felt that the fact that such infections are such a very frequent accompaniment to thyroid disease "constitutes an important factor in maintaining a condition which favors over-activity of the thyroid gland. One cannot see the effect of repeated infections in these patients without being impressed with their importance."

The writer would urge upon all nose and throat men the wisdom of making every routine examination include a careful scrutiny of the thyroid gland. While the consensus of opinion already cited seems to be that once thyroid disease is well established no amount of attention to tonsil conditions can bring about any improvement, we certainly have abundant evidence to show that a thyrotoxicosis in its initial stages may be retarded or even completely aborted by the extirpation of infected tonsils.

The suggestion contained in Dr. Poynter's reply that he believed if a relation between the tonsils and thyroid is ever demonstrated, it will be through internal secretion, opens up a wide and fascinating field of investigation. The laborers in this field are more likely to be those who are approaching the question from the thyroid side, and upon them the writer would also like to urge the necessity of minutely considering the state of the tonsils of every goiter patient presented to them for the first time. Especially in those cases where a tonsillectomy has left a certain amount of tonsillar tissue in the throat and the healed surface may still be masking infective pockets it will be well to seek the cause of the thyroid disturbance in the tonsillar region.

It is in the hope of thus stimulating interest and, if need be, arousing criticism and opposition which will result in a more widespread consideration of the entire subject, that the present effort to call attention to it has been made.

CONCLUSIONS.

The conclusions drawn by the author from the result of a canvass of practitioners whom he believed to be in a position to give information regarding the possibility of a relation between goiter and tonsil infection were:

1. That comparatively little attention had been given to a consideration of this possibility.
2. That the majority believe goiter to be largely of toxic origin.
3. The tonsil is no more likely to be the focus of infection than any other location—e. g., sinuses, teeth or gall bladder.

A survey of the scant literature relating to this subject seems to indicate that those who have investigated the coincidence of goiter and infected tonsils, and have exhaustively considered their possible interrelation, incline to the belief that diseased tonsils may in many cases be directly responsible for goiter, both simple and exophthalmic.

It is urged upon the throat specialist that he give particular attention to the state of the thyroid gland in all cases of infected tonsils, and also upon those who are called upon to treat disordered thyroids, that they bear in mind the possibility of an exciting factor in the presence of diseased tonsils.

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XXVI.

SUBMUCOUS RESECTION—COMPLICATIONS AND AFTER-RESULTS.

BY N. S. WEINBERGER, M. D.,

ROBERT PACKER HOSPITAL,

SAYRE, PA.

Submucous resection of the nasal septum, so frequently indicated in otorhinology, cannot be studied and developed too carefully. Although the essential features of the technic have become practically standardized, this paper is written with the idea of reviewing the possibly too frequent complications and discouraging after-results, and with the view of determining in what ways their occurrence can be minimized. Submucous resection is an operation that is at times unnecessarily performed, and this is to be deprecated, but it has not yet reached the point when it sufficiently and universally displaces the mutilating operations and makeshifts of former years. The fewer the complications and the better the after-results, the sooner will this end be accomplished.

The technic will not be discussed excepting in so far as it affects the considerations of the title of this paper. Speaking in general, from what one learns from the literature and from personal experience, it would seem better if submucous resection were regarded from the standpoint of surgical risk. All patients should have a routine general physical examination, including the blood and urine, as well as a very careful local examination of the throat, teeth, ears and accessory sinuses. The operation should always be done in a hospital and the patient put to bed in the upright or semireclining position until all packing has been removed. In this way hemorrhage will be very much less frequent and the postoperative congestion much lessened, thus adding a factor of comfort to that of safety.

It might be well to consider the much discussed question of packing. As far as hemorrhage is concerned, a properly

packed nose will very rarely bleed excessively. It is generally accepted that packing increases the possibility of other complications, notably the infections of the operative field, the sinuses, the ears, the meninges or the throat. The question, therefore, resolves itself into one of relative importance and expediency in which the factor of safety must be included. Granted then that it is better to pack the nose from the standpoint of hemorrhage, we are justified in packing with the increased danger of these infections, realizing that the local result will be better and the purpose of the operation more fully served, or should we take the chance of the occasional unsatisfactory result? The infection of the operative field when properly cared for does not in itself mitigate the benefits of the operation, but rather enhances them, and infection can and does at times occur when no pack is used.

We should always obtain the best results possible and should do all in our power to minimize the occurrence of infections with the pack and thereby decrease the danger to the patient. It appears that with due precaution the evidence favors the pack in spite of the temporarily added discomfort of the patient and the possibility of infection, especially so since absence of the pack is only one of the factors of non-infection.

These precautions are as follows: General physical and laboratory examination before operation; local examination of the sinuses, ears and throat, and wherever feasible the preparatory care of any infection that may be present, either by treatment or operation, with the idea of producing as clean an operative field as possible under the conditions.

Strict aseptic technic is essential. Submucous resection should not be done in the presence of an acute infection or immediately following some other local operation, however slight. If, in case of unilateral acute sinus infection, it is imperative to do a submucous resection, the operation should be done from the other side.

Patients should be put to bed in the upright position on a backrest for twenty-four hours. They should be cautioned against attempting to blow or force air through the nose and to open the mouth and cough when there is a desire to sneeze, thus avoiding great and sudden changes in postnasal and mid-

dle ear air pressure. The diet should be liquid until the pack is removed, so as to minimize the negative postnasal and middle ear pressure occasioned by swallowing. The pack should be removed in twenty-four hours, unless for some reason it is feared that repacking may be necessary. Repacking should never be done if it can be possibly avoided. Postoperative irrigation is dangerous, especially in the hands of the patient. If irrigation is employed it should be practiced with great caution, advising the patient of the importance of avoiding forcible blowing of the nose and sneezing. When the nose is packed forty-eight hours, the second twenty-four hours favors infection proportionately greater than the first twenty-four. The postoperative reaction and congestion have abated, and the active resistance of the first day has given place to the passive receptivity of more or less traumatized tissues in addition to the prolonged interference with the drainage and ventilation of the sinuses.

Then again, the improper use of the pack unfortunately adds to its disfavor. Splints and prepared tampons are not so good as gauze wicks in that they cannot be applied so as to give uniform and even pressure over the entire septum. They are more apt to slip out of place, thus allowing serum and blood to separate the flaps in circumscribed areas. This usually occurs high up, anterior to and below the middle turbinate, where infection is most liable to occur because of less vascularity in that region.

The gauze wick pack properly applied will with more regularity leave a straight thin septum, and, all other things being equal, assists in producing the utmost benefit. The flaps are held in apposition, hematoma is rare, and subsequent ballooning of the flaps from air currents and from blowing the nose is avoided. The slightly longer convalescence is nonessential. In a series of cases the disadvantages of packing seem to be outweighed by the uniformly better after-results.

Hemorrhage of any consequence usually occurs from the bony ridge below and well forward, or from the edge of the vomer well back, and has in some reported cases been alarming, notably in a case reported by Montgomery, which came on six days after operation. It is most likely to occur in the first twelve hours after operation and less likely in the second

twelve hours. If the nose is properly packed, especially in the region of the hard parts, it seldom occurs. It is well to attack the ridge below and in front last.

The infections are usually the result of faulty asepsis, infected membranes, sinuses or tonsils. They consist of abscess and perichondritis of the septum, meningitis (nine cases collected and reported by Dabney), and acute sinusitis, especially when chronic disease is present and the nose is packed too long or is repacked, acute otitis media and mastoiditis, tonsillitis, peri- and endocarditis, septicemia and cavernous sinus thrombosis (case reported by Hays).

The middle ear and mastoid infections are usually streptococcic and virulent, and often originate in the throat. Perforation should be rare and is usually due to too much haste. It results from poor elevation (not getting beneath the perichondrium), from spicules of bone and from rapid and careless operating. Perforation is unavoidable in a small percentage of cases with bad deflections, those with very sharp angles, where there is overriding and dipping in of the mucous membrane. It does no harm if the edges are free from cartilage and bone so as to allow apposition of the free edges of the mucous membrane, but its prevention should never be sacrificed for rapid work nor for cosmetic reasons alone. The removal of all deflections should not be compromised for an unavoidable perforation.

Sinking in of the nose is due to removal of too much of the cartilage of the bridge anteriorly, to improper care of post-operative infection, perichondritis and abscess and to syphilitic and diabetic necrosis.

There is a wide divergence of opinion as to the limitations of age at which submucous resection should be performed. Some operators have practically none except the very young and report a fair percentage of sunken noses in children; others do not operate after middle age nor before the facial bones have fully grown. There should be no upper age limit when ventilation and drainage of the sinuses are required. The attitude toward a lower age limit should be that of conservatism as far as possible until facial development is about complete. It is not possible to specify the lower age limit in years, because of early or late development, but, generally speaking,

the operation is contraindicated in children under fourteen who develop early. Other contraindications are untreated syphilis, diabetes, advanced tuberculosis, acute sinus disease, acute middle ear disease, atrophic rhinitis and marked hypertrophic rhinitis.

Unless all deviated portions are removed, especially those high up between the middle turbinates, those close to the floor of the nose, the ridges and spurs of the vomer well back and split cartilage or vomer, apposition of the flaps will not be good, healing not so prompt and the result is apt to be discouraging.

It is well to make drainage incisions well back to allow escape of serum and blood rather than have a collection between the flaps, which would interfere with good apposition, form a culture field for infection or leave a thickened septum or ballooning flaps. Permanent adhesions between the septum and turbinates or outer wall should not occur, as they show undue traumatism from careless use of instruments. They usually occur where, in attempting to remove a sharp angle or deviation, considerable traumatism and tearing is done to the mucous membrane of the septum and the outer wall or turbinate directly opposite, and the attempt at removing the deviation is abandoned and two abraded areas are allowed to come into apposition. Permanent adhesions should not occur in the hands of careful and thorough operators. They usually interfere with good results, or because of remaining deviation require reoperation.

The question of the anesthetic is important. Cocain and adrenalin in varying proportions and strength is the anesthetic of choice by most operators. General anesthesia is very little used in this country, and not only seems unnecessary but impractical. Subperichondrial injection of Schleich's solution has its advocates, but offers difficulties that prevent its universal adoption. The untoward effects of cocain can be minimized by preoperative general examination, operating in a semi-reclining position and avoiding excessive use of anesthetic. In this way fainting and syncope, which sometimes interfere with the progress of the operation, are practically avoided.

Cocain poisoning in susceptible patients occurs rarely and needs separate attention. Hysteria, transient hysterical paralysis, erysipelas and fracture of the cribriform plate have been

reported. MacKenzie reports a case of air embolism. An anatomically normal nondeflected septum is rare. Submucous resection should not be done because of deflection alone. We should be guided by indications and have a reasonable assurance that the patient will receive real benefit or that probable future trouble is prevented.

The operation is not entirely successful unless its interference with drainage and ventilation of the sinuses has been remedied, but when indicated and well performed often yields brilliant results.

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XXVII.

TREATMENT OF ACUTE SINUS INFECTIONS.*

BY LEE M. HURD, M. D.,

NEW YORK.

There are some cases, a fraction of 1 per cent, that require prompt and fearless surgical intervention because of the severity of the infection which has or will extend beyond the sinus limits, thereby producing grave complications.

There is a fair sized group which becomes chronic unless properly treated, but the great majority recover without attention or they undergo medical treatment or minor surgical measures.

The cardinal symptoms for which the patient seeks relief are pain, pus, pyrexia and swelling, in the order named. Many times they are treated by home remedies or by general physicians for neuralgia, or, if influenza is epidemic, the case with headache and fever is treated for influenza, even if the nose is occluded and discharging pus.

GENERAL CONSIDERATIONS.

For Pain.—Heat, electric light, diathermy or hot applications, such as poultices, are used. The electric light can be applied in the form of the Killian box, the therapeutic light or what is quite as efficient and much more simple, the ordinary droplight hung over the patient's head, so that they may get heat and light at will. Drugs—Aspirin, the coal tar derivatives and opium derivatives are prescribed.

For Congestion.—Cocain is highly recommended, but it really is irritating and extremely dangerous to give the patient as a spray; adrenalin, also is highly recommended, but its benefits are counteracted by its reaction; nitrate of silver in small doses of $\frac{1}{2}$ to 1 per cent as a local application, after the first slight irritation, gives more lasting contraction than either

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cocain or adrenalin; argyrol of doubtful service, and hot saline douches. Of the four agents I depend mainly on the saline douche and nitrate of silver.

For Pyrexia and External Swelling.—Surgical relief by drainage.

A rough working rule is that if the three symptoms—pain, fever and external swelling—are present, the case is beyond medical measures and requires prompt surgical intervention. This, like all working rules, has its exceptions. But if the infected process has extended beyond the sinus limits, usually into or about the orbit, producing cellulitis, abscess, caries, osteomyelitis, ocular symptoms or cavernous sinus symptoms, though these conditions are very rare, they require prompt external operations for the relief of pressure and drainage of the sinus and the infected area.

TREATMENT DIRECTED TO PARTICULAR SINUSES.

Acute Ethmoiditis.—The medical treatment outlined above under general considerations meets the requirements of acute ethmoiditis and very rarely indeed is surgery indicated. This may be required by an extension of the infection through the sinus walls into the orbit or threatening the anterior cranial fossa, which may call for an external operation by the orbital route.

ACUTE FRONTAL SINUS INFECTION.

The usual medical measures to reduce the congestion within the nose, with suction to reduce the amount of pressure within the sinus, will usually suffice. If there is the slightest evidence of swelling of the lids or an elevation of temperature it becomes necessary to remove the anterior portion of the middle turbinate and break down the anterior ethmoid cells which form part of the floor of the frontal, to establish drainage. It is possible only in a few cases to catheterize the frontal sinus without this surgical measure. If it does not promptly relieve the temperature and swelling, it becomes necessary to invade the sinus through the eyebrow, but this is extremely infrequent and it is done to relieve such conditions as orbital cellulitis, osteomyelitis and caries.

There is one type of acute frontal sinus which is characterized by a periodic neuralgic pain which comes on at nearly the

same time in the morning and as suddenly ceases about the same time in the afternoon, and the patient feels quite well from that time until the next morning. I have no satisfactory explanation for this type; though they suffer the most pain, they are not inclined to be as serious as the more common type.

If the pain is not too severe and does not extend over too many days, I try, by the aid of local depletion, suction and diathermic heat externally, to evacuate the secretion, which is of a thick mucoid character, rather than purulent, because as soon as this thick collection is removed from the sinus the pain disappears. I have demonstrated that in several severe cases, by enlargement of the frontal orifice and douching out this mucoid plug, the pain promptly disappears.

The sphenoid sinus, besides the general measures, can usually be catheterized and the contents douched out. If it is impossible to catheterize, I depend upon suction. If suction fails to give relief, the severe pain indicating much pressure, I take down the anterior sphenoid wall.

ACUTE ANTRAL DISEASE.

A very few are of dental origin. This can be relieved only by the removal of the offending tooth. The antrum should be douched and if possible the antral cavity should not be connected with the mouth. If it does, the tooth socket should be packed with iodoform gauze.

The vast majority of cases are due to extension of the infection from the nasal cavity. For the pain, electric light or diathermic heat should be applied, local depletion and suction, with the head on a horizontal plane and the affected antrum uppermost. But the best method is to douche the sinus with saline through the normal orifice if possible, otherwise by needle puncture through the inferior meatus.

The washings from an extremely acute sinus in the very early stages will be clear to cloudy or bloody serum. This may abort the infection from going on to a frank pus, but usually by the time the patient consults the physician the antrum contains pure pus or mucopus. The antrum should be douched according to the severity of the infection, every day or every other day.

Diathermic heat should be applied over the frontal or antrum, as the case may be, in the form of a small metal plate about the size of the sinus, and another larger plate on the back of the neck, and the current should be used up to the tolerance of the individual with the heat created, without burning the skin. This will actually raise the temperature within the sinus and, if used from five to ten minutes, will relieve the pain.

Suction is much under controversy as to its value. There should be no discussion as to its merits for diagnosis, and it is often a valuable adjunct to treatment. In fact, it should be routinely used in acute infections of the sinuses where it is impossible to catheterize and douche them out. It is necessary to control the amount of vacuum used by a reliable gauge, usually between 10 and 15 inches being employed, or less if the patient cannot tolerate it. This should be reduced by a proper tip in one nostril, the other nostril closed and the nasopharynx shut off, having the patient say k, k, k. The vacuum should not be continuous, but should be on for a second or two and then broken, and rapidly repeating this procedure a number of times or, in fact, as long as the suction produces pus. It is well to keep the suction down to a point below which it extravasates blood.

XXVIII.

SOME FURTHER OBSERVATIONS ON THE ETIOLOGY AND TREATMENT OF MAXILLARY SINUSITIS.*

BY HOWARD V. DUTROW, M. D.,

DAYTON, OHIO.

It is interesting to note, in reviewing the literature, the great diversity of opinion as to the etiology and treatment of maxillary sinusitis, but upon close analysis it is evident that steady progress is being made and something approximating standardization achieved. No doubt, the demand for more knowledge regarding the diagnosis and treatment of infections of the nasal accessory sinuses in general has been actuated by the indifferent end results obtained by many good workers in this field. Hence the voluminous amount of literature and the many conservative and radical surgical procedures elaborated to meet all classes of cases.

It has been our too frequent experience in years gone by to operate two or three times upon a case of chronic maxillary sinusitis, and even then to find results disappointing. This condition has improved in that most of us are now giving much more study and attention to diagnostic and operative refinements and are consequently getting better results.

Heretofore most of us were inclined to regard the antrum as a reservoir into which pus drained from infected frontal and sphenoid sinuses and from the ethmoid cells. I am confident that the ratio of the source of infection has to be reversed. Surely more infections are now known to be due to infected teeth and are commonly termed ascending infections, as compared to those coming from above, or descending infections. The percentage due to the teeth has been variously estimated from as low as 8 per cent to as high as 100 per cent. Two yaers ago I stated in a paper read at Kansas City, that from

*Read before the Ear, Nose and Throat Section of the Ohio State Medical Association at Cincinnati, May 4, 1922.

65 to 70 per cent were of the ascending type. Some have felt that the percentages quoted were entirely too high, but I am confident that these same men now would consider them more nearly correct. It is very seldom that we find a chronic maxillary sinusitis in a case where the dental line is unbroken. If the teeth are examined and a careful history regarding them obtained it will be found, in a great majority of cases, that a first molar tooth, more frequently, and very often the second molar tooth, or both, have been extracted. Many crowned or filled molar teeth whose pulp and nerve supply has been killed will also be noted.

Freer, of Chicago, has recently stated that it is his belief that practically all sinus infections were of the ascending type. While his estimate may be too high, I believe it more nearly correct than the estimate of those who claim a maximum of only 8 per cent ascending infections.

To prove further my conclusions as to the preponderance of the ascending over the descending infections, I recently had a case of chronic empyema of the left sphenoid sinus, which was of fifteen years' duration. In making a diagnosis of this case by process of elimination, transillumination and X-ray findings, the antra were negative, but the sphenoid sinus showed a definite empyema; the antrum was irrigated through the ostium and the fluid was returned perfectly clear. The ethmoid labyrinth had been partially exenterated fifteen years previously, but the sphenoid sinus was not suspected at that time. Here was a case of pus pouring out of the ostium of the sphenoid for fifteen years, yet there was no infection of the maxillary sinus.

Another case to support my belief as to the source of infection was one of chronic maxillary empyema, in which the first and the second upper molar teeth on the same side had been extracted some twelve or fifteen years previously, and for which a rhinologist some eight or ten years ago removed some nasal polypi, but did not do a middle turbinectomy. A Caldwell-Luc operation was done and the sinus found to be filled with granulations, some of which were protruding through the ostium into the middle meatus. There was some fetid discharge from the sinus for about a week or ten days following the operation, which had to be washed out. Improvement was noticeable

after each succeeding irrigation. The discharge was rather slow in disappearing, and it was felt that, according to the generally accepted belief, there might be some drainage from above into the antrum. An ethmoid exenteration was done, for which I have my own personal regrets, because there was absolutely no pathologic condition within the ethmoid labyrinth. In fact, the morning I had selected for the ethmoid operation I debated as to whether or not it should be done, because the discharge from the sinus was practically absent. I here wish to caution against too much haste in destroying the middle turbinate and ethmoid cells, if there is still some discharge following a Caldwell-Luc operation. A reasonable length of time should elapse in the hope of saving the intranasal structures.

During the past several years I have had many cases in which, upon examination of the nose, I found the middle turbinate and ethmoid region bathed in pus, with some polypoid degeneration of the mucous membrane, which was in constant contact with this secretion, to clear up entirely and remain normal after a Caldwell-Luc operation.

I believe that an infected maxillary sinus is far more likely to infect secondarily the ethmoid labyrinth and even the frontal and sphenoid sinuses, rather than the reverse.

Most of us forget the fact that we occupy the horizontal position eight to twelve hours out of every twenty-four, and it is at this time that the head is very often somewhat lower than the plane of the body, and infectious material can easily find its way into contiguous sinuses and cells, thus producing a secondary infection. The middle turbinate normally forms a complete cap over the ostium, and anatomically it is difficult for secretions to enter the sinus through the rather slitlike or oblong ostium.

I have not had a case of reinfection of the maxillary sinus to occur following a Caldwell-Luc operation. In more than 90 per cent of these cases no intranasal structures received any surgical interference at my hands whatsoever. Some had had some intranasal work done previously by other men.

This is a point, I believe, worthy of serious consideration, because, if the principal source of infection is from above, nothing has been done to prevent a subsequent infection. My

remarks are not to be construed as indicating that infection from above is not possible, but it is very rare, and therefore the structures of the upper part of the nose should be saved in every way possible. The permanent anatomic and physiologic damage done by a well performed Caldwell-Luc operation is negligible as compared to that of a destructive intranasal operation.

The old theory of Jansen that if one sinus is infected, all of the same side would be infected, is obsolete and, I believe, thoroughly disproved. To open all of the sinuses on one side because one happened to be infected is meddlesome surgery. Much time and care should be given the question of diagnosis to determine as accurately as possible which sinus or sinuses are involved. The old shotgun diagnosis is a thing of the past, because we now know that the infection may be limited not only to one sinus but even to a compartment or one-half of a sinus.

My end-results in some forty-odd Caldwell-Luc operations performed during the past three or four years have been eminently satisfactory from every standpoint. Prior to that time I was rather discouraged with my treatment of chronic antrum infections, but after careful study and strict attention to operative detail better results were obtained.

Much has been said regarding the loss of nerve supply to the incisor and canine teeth of the operated side. I have had only one case in which this sensibility was permanently lost, and I am quite sure that in this case I made my opening too low and severed the nerve supply. Some men have taken the graphic nerve distribution to these teeth, as shown in Gray's Anatomy, as correct. I believe this graph was made for purposes of illustration only. The nerves are shown as coming off in a fan shaped distribution from above downward, from the infraorbital foramen, and if this is true we would have loss of sensibility in the teeth so supplied in every case operated upon. As a matter of fact, the nerve supply goes almost vertically downward, just posterior to the canine fossa, and comes forward in the dental foramen. If the opening is properly placed as near the center of the canine fossa as possible and enlarged peripherally, care being taken not to carry the re-

moval of bone too far downward, this complication need not arise.

I am sure that if most of us will compare our technic of operation of today with that of three or four years ago, we will not be at a loss to account for our more successful treatment at the present time. Now we make an effort to see clearly all of the ramifications of the sinus and curette firmly but gently all visible pathology. In this curettement I am not sure that all of the mucous membrane is removed, certainly none of the periosteum if healthy should be removed. But it is absolutely necessary to get out all granulating tissue, no matter where it may be found. The ostium should be carefully inspected and all granulations removed. The concavity which extends into the malar bone is a very frequent hiding place for a large granuloma, as well as above and posteriorly, just beneath the orbit and in front in the angle above the incisor teeth. The slogan of all radical surgical procedures in sinus work should be absolute thoroughness in removing diseased tissue. The nasal opening should be carefully and skillfully made and a flap preserved and made to lie upon the floor of the sinus, thus covering the angle of bone which formed the nasoastral wall of the inferior meatus. It is an anatomic impossibility to have the floor of the nasal fossa on a level with the floor of the antrum, as in practically all cases the floor of the antrum is from one to three or four millimeters below the floor of the nose. This, however, is immaterial for ample drainage, because of the many changes in the posture of the body.

Another great permanent advantage of a good sized nasal opening is the constant aeration of the sinus. It is known that for granuloma to grow or thrive, they must be protected from the air. This, I think, explains why, if a large enough opening is made in the inferior meatus, one that cannot grow shut, these cases remain well. It may be argued that the sinus under normal conditions is filled with air. This may be true, but the ingress and egress must be through the ostium, which may be closed for long intervals, depending upon the congestion of the nasal mucosa, whereas, if the opening in the inferior meatus is large enough, the air is constantly changing.

Irrigations should be used with great discrimination. There are cases where they are positively beneficial for a short time or for as long as they may seem to be needed, but they should be discontinued as soon as the sinus approximates normal.

There need be no argument that the radical surgical treatment is best adapted to the chronic infections. There are undoubtedly cases of acute antrum infection, the source of which may be through the blood stream or by continuity from the nasal mucosa, or through the lymphatics, and it is in these cases, if seen early, that puncture and irrigation will cure. But if the case is of long standing, it is like dealing with an unknown quantity to rely upon puncture and irrigation. You are groping in the dark.

To illustrate, I will cite briefly an observation. A child ten years old, with a chronic right maxillary sinusitis. The skiagraph showed unerupted teeth well up in the canine fossa, and it was considered inadvisable to do a Caldwell-Luc operation. This little patient was operated upon through the inferior meatus. A large opening was made and as much of the polypoid contents of the antrum removed as possible. The operation was done some six or seven months ago, and the child has had to be irrigated at least once a week ever since. She has made slow but steady progress, and the discharge now is very little. She has gained considerable in general health, but I am not satisfied with the interior of the sinus, because I am confident that it is at least partially filled, and will remain so, with granuloma. The ciliated epithelium, which some authors claim to do a great deal in wafting secretions toward the ostium, do not seem to have accomplished very much in this case.

The improvement in general health of the cases operated upon for antrum infection of long standing is most remarkable. These patients gain in weight, sleep well and the vague neuritic and myalgic pains disappear and they are very much benefited in every way.

CONCLUSIONS.

We should not be carried away by any one method of treatment to the exclusion of all others, but cases should be thoroughly studied and the procedure adopted that is going to give

the best complete and permanent results.

1. Definite progress is being made in the diagnosis and treatment of maxillary sinusitis.

2. Observations indicate that our earlier ideas as to the etiology and sources of infection have to be revised.

3. Destructive intranasal surgery should never be resorted to until after the diseased antrum has been treated and sufficient time has elapsed for the structures within the nose to return to normal.

4. No reinfections have occurred following a Caldwell-Luc operation, in which the middle turbinate and ethmoid labyrinth remained intact, thus disproving to a great degree the theory of descending infections.

5. In chronic empyemas with granuloma, absolute thoroughness in removing all pathologic conditions within the sinus, together with adequate drainage and constant ventilation, are three cardinal points necessary in the satisfactory treatment of these cases.

6. End-results as to absence of deformity or destruction of physiologic structures and the greatly improved condition of the patient fully justify proper radical surgical intervention in this class of sinus infection.

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XXIX.

THE UPPER RESPIRATORY TRACT IN GRANITE DUST INHALATION.*

By D. C. JARVIS, M. D.,

BARRE, VT.

Vermont having the largest granite center in the world, the opportunity to study the effects of granite dust inhalation upon the respiratory tract is exceptional. A permanent residence rules out the time factor and makes it possible to carry the work to any conclusion thought advisable. Observing the upper respiratory tract of granite cutters for the past twelve years, one's attention could not fail to be focused sooner or later upon their difficulties, and in 1917 a definite effort was made to observe in detail the condition these men manifested.

Introduction.—The accumulation of cases was slow, and the advent of the National Tuberculosis Association in the field during 1919 was welcomed. Through its Committee on Mortality from Tuberculosis in the Dusty Trades, an investigation of the granite industry was started because it was thought that the granite industry, of all the dusty trades, would show the highest mortality from tuberculosis. The investigation was divided into three phases: A statistical inquiry, a mechanical process inquiry and the medical examinations. The granite cutters have shown a remarkable spirit of cooperation. They did not appear because of any illness but came up for examination in routine order by previous arrangement. This previous arrangement consisted in taking the subject up with the Executive Committee of the local union, which in turn called a meeting of the men for a consideration of the subject and action. Many of them had never had occasion to consult a physician before. They were all able to work and considered themselves in good health. At this time it is possible to report on the examination of 500 granite cutters.

Review of the Literature.—While numerous references are

*Candidate's thesis. American Laryngological, Rhinological and Otological Society.

found in the literature to dust inhalation in general and some forms in particular, from Ramazzini's article in 1700 down to the present time, one fails to find a record of a definite study of the effects upon the upper respiratory tract of granite dust inhalation. The term "stone cutters" is found, but with no particular effort to separate granite cutters from marble workers and stone masons. The general report of the Miners' Phthisis Prevention Committee of South Africa, published under date of March 15, 1916, comes the nearest to the present work, but their similarity lies in the composition of the stone and not in the manner of handling. The South African process I understand to be a blasting one, while the Vermont process is in finishing the stone for monumental purposes, the stone being worn down by means of a pneumatic tool. The work in Vermont represents the first work of the Committee on Mortality from Tuberculosis in the Dusty Trades. The only other investigation of a dusty trade in this country is represented by the valuable contribution to be found in United States Bulletin No. 85, issued January, 1917, comprising a clinical study of 433 cases of miners' consumption among zinc miners in Southwestern Missouri, by A. J. Lanza.

Experimental Pathology.—Dr. L. U. Gardner of the Saranac Laboratory is in charge of the experimental pathology connected with the investigation, and the reader is referred to his article in the December, 1920, number of the American Review of Tuberculosis for the preliminary details of this phase of the work. One specimen from the upper respiratory tract examined by Dr. Gardner was reported as follows: "Examination of tissue from granite cutter's nose of September 25th shows a mucous polyp composed of a mucous connective tissue stroma covered with columnar epithelium on the surface. Mass contains frequent gland acini and in places infiltrated with plasma cells. No trace of granite dust nor any reaction suggesting this irritant."

Evidence on Which Report Is Based.—The economic phase was so important that attention was paid to small details, such as the presence of a mustache, its growth as to luxuriance, the length of time it had been worn, and the amount of hair in the nose; the condition of the septa, the turbinates; the presence of lymphoid tissue upon the posterior pharyngeal wall; the

condition of the tonsils, teeth and the larynx. From the observations necessary in accumulating this evidence the deductions and conclusions made in this thesis have been derived.

COMPOSITION OF GRANITE REPRESENTED BY THIS STUDY.

Silicon dioxid.....	69.56
Aluminum oxid	15.38
Iron oxid.....	2.65
Magnesia oxid	Trace
Calcium oxid	1.76
Sodium oxid.....	5.38
Potassium oxid.....	4.31
Manganese	Trace
Loss in ignition, CO ₂ and moisture.....	1.02
	100.06

According to Gardner, when examined under the microscope the unscreened dust may be roughly divided into large and small particles. The former, which could not possibly be concerned in an inhalation experiment, show an average size of 82 x 84 microns. The smaller particles average 4.3 x 6.3 microns by actual measurement. In shape the particles may vary greatly. Some are flattened polygonal masses with sharply cut edges and corners. Others are rod shaped, often slightly curved with sharply horizontal or square cut ends. Frequent vegetable cells and fibers are noted. Samples of dust resulting from the use of the pneumatic tool have been examined by means of a small giant magnet made by the Victor X-ray Corporation and considerable steel has been extracted from a given quantity by means of the magnet, 2 drams of granite dust yielding 2 grains of steel. From this experiment it will be seen that we are dealing with two kinds of dust, that from the stone being worked and that from the tool used in working it.

Present Problem.—In 1915 there were 2050 granite cutters working, while in 1919 only 1240. The question suggesting itself, whether this condition was peculiar to Vermont alone, the total number of cutters for the United States was investigated and it was found that there was a corresponding diminution in the number of cutters throughout the whole country. The statistician returned the report that an analysis of the

death certificates for the past twenty years indicated that 86 per cent of the cutters died from tuberculosis. The present study was undertaken to determine if possible the part played by the nose and throat in the developing of the clinical condition. A study of the industry reveals men who have inhaled granite dust with impunity over periods ranging from thirty to fifty-nine years, and other men who are punished severely by granite dust inhalation even when less than fifteen years represents their period of exposure. Why do some men suffer severely from the effects of dust inhalation, and why do others escape so easily?

The Occupation in Itself.—An occupation that is dusty does not justify the conclusion that affections of the respiratory tract are prevalent to a large degree. Also if an individual working in a dusty trade develops an affection of the respiratory tract, dust inhalation may not be the cause. There are other factors, such as hygienic conditions, to be considered, and while a close study of granite dust inhalation reveals the fact that a certain proportion of the men have an affection of the respiratory tract superimposed. One who works among these men daily cannot help but feel that a vicious circle is developed; that the inhalation of granite dust in itself is not a producer of a serious respiratory disease, but rather produces other conditions of the respiratory tract less serious, such as frequent colds, bronchitis and pneumonia, which in turn prepare the respiratory tract for the tuberculous process.

Suitable Soil. As one works longer among these men, there comes a feeling that certain ones represent suitable soil for the early manifestation of the injurious effects of granite dust inhalation. Knowing that all men were not safe in the industry, and that some broke down earlier than others, it became necessary from an economic standpoint to ascertain if possible the type of individual who would not last long in the industry under present working conditions, and to suggest that he take up some other trade. While the period of apprenticeship is three years, it takes between eight and nine years to bring a cutter to a final degree of excellence. Although these men have been studied during the past twelve years, it is only during the past five years that an effort has been made to discover the type of individual who might be accepted as an apprentice.

This type represents a hazard which, if accepted, would soon break down in the industry and represent an economic loss to both industry and state. It seemed advisable to first examine the survivors in the industry to ascertain if possible the individual characteristics they possessed and their mode of life. Accordingly cutters who had inhaled granite dust as long as fifty-nine years were examined and detailed data kept of their condition. As stated before, the economic phase was so important that attention was paid to small details. Cutters who had broken down were examined and also several hundred cutters who were working all the time, endeavoring to find the type of individual who would last reasonably long in the industry. As a result of all this work, we are able to tell the type of individual representing a hazard, and keep him out of the industry and select the individual who would do well. It was found that the upper respiratory tract held the key to the situation and that an individual was a good risk in proportion to the absence of lymphoid tissue in this region. An apprentice with normal turbinates, a smooth posterior pharyngeal wall, tonsils small in size, no evidence externally of enlarged lymph glands, represented a desirable type and one who under ordinary conditions would last in the industry. On the other hand, if a young man 20 years of age showed hypertrophic rhinitis, enlarged tonsils and lymphoid nodules on the posterior pharyngeal wall he should keep away from the industry, as he will break down early under present working conditions without dust removing devices. On the basis of suitable soil, it was found that the different nationalities lined up in a definite manner. In order of excellence they were found to line up somewhat as follows: Italians, Americans, English, Spaniards, Scotch, Swedes, Norwegians, Danes, French and Irish. You will notice that there is a geographic distribution of these races, many of those being least affected by the dust, such as the Italians, living in Southern Europe, and many of those along the last of the list living in Northern Europe. They may be also classified on the basis of those living in dusty countries and those living in countries not so dusty. It cannot be denied that, in the granite industry at least, the Irish are punished severely, and it is a question whether they should be in a dusty trade, certainly not in the granite industry, as they

represent a hazard, under present working conditions, and should not be accepted as apprentices.

Suitable Preparation of Soil.—As a result of granite dust inhalation there occurs an adaptation to occupation of the upper respiratory tract which may be estimated in various ways. If a pencil point is drawn across the posterior pharyngeal wall of a cutter he will not know by the feeling that anything has touched his pharyngeal wall. A laryngeal mirror may be held against the soft palate indefinitely, and the cutter states that he feels nothing in his throat. The ordinary gargles are tasteless to him and he recognizes them only when they are very hot; all throat applications must be increased in strength.

The adaptation to occupation is best studied in connection with the eye, for here one is able to remove foreign bodies from the cornea without the aid of cocain, and these may be present without the usual accompanying signs of irritation. Small pieces of granite 2 to 3 mm. in length have been observed floating about in the conjunctival sac without the cutter knowing of the presence of a foreign body. This adaptation to occupation apparently is a defense whereby dust inhalation is tolerated. While, however, it acts as a defense during continued work it acts as a detriment when absence from work results for any great length of time, because in proportion to the length of time a cutter remains away from work does he lose his adaptation to occupation. It is a well known fact that if a cutter is absent from work one month, on his return for the first three or four days he expectorates a great deal of dust, which comes up in little round balls, moist on the outside, but when rolled between the thumb and finger the center of these little granite balls is found to be dry. With the passing of three or four days this is discontinued, as adaptation has again occurred. With longer periods of idleness this adaptation to occupation is not so easily regained. Cutters themselves have discovered that something happens as a result of these periods of idleness brought on by strikes, lockouts or poor business, because they have observed and so state that a resumption of work following one of these periods of idleness causes the breakdown of many men. Granite dust inhalation in itself is not the direct cause of tuberculosis, but it is the suitable preparation of the soil when a cutter is en-

deavoring to reacquire his adaptation to occupation that assists in spelling his downfall.

Here then we have a vicious circle, granite dust inhalation requiring adaptation to occupation of the respiratory tract, absence from occupation causing him to lose this adaptation and a reentry into the industry producing irritation of the respiratory tract while he is endeavoring to regain his lost adaptation. This last effort brings about a suitable preparation of the soil. With two hundred dusty trades listed in the United States, one wonders how much this adaptation, lost and regained many times during a worker's occupational life, is a factor in the production of disease of the respiratory tract. Certain it is that if a cutter absents himself five years he may return if his initial exposure has been less than ten years, but any cutter who has been absent from the industry two years should think twice before reentering, because the difficulty in regaining adaptation to occupation represents a handicap too great for many to overcome, and suitable preparation of the soil takes place, with the resulting development of a tuberculous process.

Mouth Breathing.—According to the experience of Watt Irving, Johnson and Stewart, in the South African investigation, the mouth breather is more apt to suffer from the effects of granite dust inhalation than one who employs nasal breathing. Particular interest has been taken in this phase of this subject, and Roentgen ray films of cutters showing mouth breathing were separated from those showing nasal breathing, and a study was made, comparing them with other films of the same exposure period. The result shows that the films belonging to those breathing through the mouth show less lung lesion than those breathing through the nose. The explanation is that the usual curve of the inspired air is broken by mouth breathing, and the dust impinging upon the posterior pharyngeal wall is expectorated as soon as the accumulation warrants or is swallowed, with the result that very little reaches the trachea, and the lungs are consequently spared.

The Nose.—While the septum showed deviation in a number of cases, the turbinates did not give evidence of hypertrophy, apparently adaptation to occupation taking place readily. It would seem from this study that inhalation of a dust as irritating as granite does not exert a marked influence upon the

size of the turbinates, the causal factors producing hypertrophic rhinitis coming from within rather than without the body. The wearing of a mustache or the amount of hair in each vestibule did not seem to exert an influence upon the appearance of the roentgen ray films.

Diet in Relation to Lymphoid Tissue.—The diet was investigated, and it was possible to study ten nationalities in this respect, with the result that the evidence showed that the more greasy the diet the less amount of lymphoid tissue there seemed to be in the upper respiratory tract.

Practical Application of the Evidence Gained From This Study.—1. In view of the fact that adaptation of the respiratory tract to occupation occurs in dusty trades, when bronchoscopy becomes necessary, the patient's occupational history should assist in deciding upon general, local or absence of anesthesia. It would seem that, in a granite cutter at least, the passage of the bronchoscope might be accomplished without the aid of an anesthetic.

2. An individual it would seem is susceptible to affections of the upper respiratory tract in proportion to the amount of lymphoid tissue present.

3. When an individual contracts tuberculosis, assistance in its prognosis may be gained by studying the lymphoid tissue of the upper respiratory tract, the prognosis being better in proportion to the absence of lymphoid activity in this region.

SUMMARY.

1. The evidence tends to show that there exists in certain individuals suitable soil for the development of respiratory diseases.

2. In the worker in a dusty trade, adaptation of the respiratory tract lost and regained many times often acts as suitable preparation for developing a serious respiratory condition.

3. Mouth breathing is not as injurious as nasal respiration in granite dust inhalation.

4. An irritant such as granite dust from without does not seem to produce an increase in size of lymphoid tissue in the upper respiratory tract.

5. An inquiry as to the occupational history may assist in deciding the method of anesthesia when contemplating work upon the respiratory tract.

XXX.

PULMONARY ABSCESS FOLLOWING NOSE AND THROAT SURGERY.

BY JOSEPH PRENN, M. D.,

BOSTON.

In the A. M. A. Journal of August 12, 1922, Dr. Chipman and many other colleagues discuss the cause and prevention of lung abscess following nose and throat surgery.

We may state right from the outset that this complication is not a frequent one. It is rather unusual. If I remember rightly, at the A. M. A. convention held in Boston in 1921, when a similar discussion took place, one doctor cited 3,000 consecutive tonsillectomies without any such complication. Still it does happen occasionally.

The prevailing opinion is that the lung abscess is due to aspiration of infected material and that it is not likely to be of embolic origin; that when such a complication does occur it is due to some faulty technic somewhere, before, during or after operation. This faulty technic might, however, be unavoidable at the time.

If the patient has infected sinuses with an overflow of pus in the nasal chambers and nasopharynx, or if the pus appears on deep inspiration, or the patient has a bad nasopharynx, it is well to clear out the nares and nasopharynx with some medicinal application just before administering the ether.

As to the *modus operandi* during etherization and operation.

Opinions vary as to the preference of deep and light narcosis in nose and throat surgery. The advocates of one claim that the other may be responsible for lung abscess complications.

Those who are for light anesthesia reason that as long as the mucous membrane reflexes are not abolished, the patient will cough up anything aspirated, and thus prevent the infected material from lodging in the lungs.

We may call this method a second prevention. For once the patient has to cough up, then he has already aspirated, fluid has gone through his glottis and found its way in the larynx or bronchi. After coughing there is also usually a deep inspiration.

Under light anesthesia there is more bleeding, as a vasodilatation takes place.

Under deep anesthesia we simply mean to put the patient "under" so that the reflexes are abolished and the operator is not required to pause too often in his work. There is less bleeding and he can do his work more neatly and speedily. Of course, in certain selective cases the operation can be done under primary anesthesia.

The prime factor in prevention is not to allow infected material or blood to be aspirated.

When there is a great deal of bleeding, when the oropharynx is filled with blood, there is enough for the stomach and lungs. Hence, this part has to be minimized. Clamping of goodly sized cut vessels, when present, after enucleation is very important. Not alone does this afford us a clean field, but it also helps to prevent absorption from the nasopharynx through these open vessels. That is probably the reason that some patients who are operated on tonsils for rheumatism get a reaction after the operation and feel worse for a time.

The surgeon who is doing a thyroidectomy clamps the thyroid and closes all the bleeding points, not alone for the sake of leaving a clean field, but also and perhaps mainly to prevent absorption. (This procedure will serve well to obviate lung abscess of embolic origin.)

Then comes the position of the patient. Some etherize the patient in a sitting position. It is very good in many cases, when the heart is not involved. It affords good drainage, especially when there is sinus involvement.

During the operation the surgeon who sits in front of the patient can direct the etherizer when to lower the head forward to drain out secretions and, with proper amount of sponging out, can keep the throat clean so as not to be annoyed when enucleating the other tonsil.

Dr. Chipman asserts that "the right lung is involved much more frequently than the left because the right lung is larger and because the right bronchus is larger and straighter than the left, so that the entrance of infected material in the right lung is more direct and easier than in the left lung."

We must appreciate the anatomic variation of the two lungs as to their size. The right lung, though "shorter, is larger, of more capacity, and greater weight."

Infected material and blood by aspiration is therefore more apt to be drawn in by the larger lung, which is the right. Imagine two bellows of different sizes connected by one pipe having a widened out upper end, funnel like. The larger bellows is the one to draw in with greater force, hence greater velocity and solids and viscid fluid will be more apt to be sucked in by the larger one.

Added to the above we must consider the fact that it is customary for the throat surgeon to operate on the right tonsil first. Then the force from above (the source of the bleeding vessel), the gravitation and the greater suction (velocity) are all on the right side. Even though the infected material must pass through the narrow glottis, we still might consider the first two factors as adjuvants to the third one, velocity.

So when the right side has not been taken care of well; and the somewhat necessary change of position of the head more to the upright or slightly tilted backward while enucleating the left tonsil, a right lung abscess may ensue.

Then comes the postoperative stage. The position of the body and head as to drainage is essential. Otherwise the care is the same as for any patient coming out of ether.

One more point we must consider in the causation, or at least the predisposition, of this complication: The condition of the lungs of the patient at the time and also previous to the operation. When trouble is suspected we must investigate it, whether there is or recently was some existing tuberculous focus.

History, physical examination and X-ray may help.

The writer remembers a case of a young man on whom he had done a submucous resection to relieve nasal obstruction.

The patient was ambitious, but became easily tired and exhibited a lack of vitality. A negative history was given, concealing for some reason the fact (as has been revealed later) that he had hemoptysis about a year previous.

The usual physical examination before administering the ether proved to be negative.

The patient luckily made an uneventful recovery.

I heard, however, that the patient died about a year later from pneumonia, following tonsillectomy.

XXXI.

INFECTION OF THE ACCESSORY SINUSES IN CHILDREN, WITH REPORT OF CASES.*

BY EDWARD A. LOOPER, M. D.,

BALTIMORE.

During the past few years a great deal of attention has been directed to the relationship of infected tonsils and adenoids to diseases in the upper respiratory tract. So many articles have been written on this subject and so much investigation has been concentrated on these structures as a focal point for the entrance of organisms that we at times lose sight of the fact that there are other areas in this vicinity which may be playing an important role in the etiology of the disease. This applies particularly to children who have a primary or associated involvement of the accessory sinuses.

It is a common practice to attribute all chronic colds and all cases with nasal discharge, regardless of location, character or amount of secretion, to diseases of the tonsils and adenoids. It is often taken for granted that this is the only factor to be considered, while in fact the true source of the infection may be in the paranasal sinuses.

The importance of investigating this field is impressed upon us when we frequently encounter children who have had their tonsils and adenoids removed, but who do not get the desired relief of their symptoms. They give a history of colds being as frequent and the nasal discharge as constant and as severe as before operation. The operation is frequently considered as an unjustifiable procedure, the results being unsatisfactory to the patient and embarrassing to the surgeon.

A situation of this kind can be avoided if more attention is paid to the sinuses before the tonsils and adenoids are removed. In all cases of children with inflammatory lesions around the nose and throat, where the nasal symptoms predominate, the sinuses should be examined.

*Candidate's thesis: American Laryngological, Rhinological and Otological Society.

Where there is a great deal of sneezing, congestion of the turbinates, constant colds with thick profuse nasal discharge, a careful investigation of the sinuses is called for.

If the diagnosis is correctly made, treatment can be properly instituted and the results will be satisfactory.

While children are not as susceptible to diseases of the sinuses as adults, the condition is frequent enough to require careful consideration. In fact, the subject requires more attention than is generally given to it by rhinologists.

Haike¹ made an autopsy study of 394 cases of children. He opened the sinuses of 62 cases and found 52 of this number diseased. The ages were from nine months to thirteen years. Forty-seven cases had an infection of the maxillary antrum, two of the sphenoids and three had an involvement of the ethmoid cells.

Dean and Armstrong² investigated the sinuses in a group of children presenting the common symptoms of infected tonsils and adenoids. One hundred and forty-five cases were examined, and of this number 65 showed some definite involvement of the sinuses.

In a similar group of cases White³ made X-ray studies of 50 children who had been admitted to the hospital for tonsillectomy and adenoidectomy, and out of the 50 cases 41 showed pathologic sinuses.

Apparently one reason why this condition is not suspected more frequently is because there is still a prevalent opinion that the sinuses in children are so undeveloped that they do not play an important part in the etiology of early nasal infections.

In an effort to clarify this misconception, I will briefly mention our present knowledge of the anatomic construction of the sinuses in infants and children.

Considerable work along this line has been accomplished by Onodi,⁴ who has made an extensive anatomic study of 102 skulls. The material was obtained from fetuses six and a half to eight months, from the newborn, and from children one to nineteen years of age.

Since his work our knowledge of this subject has been further supplemented by the valuable investigations of Davis,⁵ and more recently by the contributions of Schaeffer.⁶

The general opinion is that the frontal sinus makes its appearance from the end of the first year to the beginning of the third year.⁷ It develops slowly and is about the size of a pea between the sixth and seventh year. From the seventh to the ninth year it is recognized as a distinct cavity.

The maximum development is reached during the nineteenth year, when it measures from 16 to 21 mm. in height and from 21 to 24 mm. in width.⁸

The sphenoid sinus is merely a faint depression in the body of the sphenoid bone at birth. Its real development begins at the end of the fourth month and generally increases in size until the sixteenth year.

The ethmoid cells are present in the newborn, as shown by Curran,⁹ who dissected fetuses from three and one-half months to birth. They develop in size with advancing years until puberty.

The maxillary antrum is most frequently infected in the child, as it has the earliest and greatest development.

According to Gegenbaur,¹⁰ "The first rudiment of the maxillary antrum is the first to appear, being seen even before the middle of fetal life, but it does not attain its complete development until comparatively late, nor does it show any considerable growth before the second year of childhood."

At birth it occupies a small space to the inner side of the orbit.¹¹ The subsequent development is downward, assuming its full shape after the eruption of the permanent teeth. The maximum development is attained between the fifteenth and eighteenth years.

So we find the maxillary antra and ethmoid cells present at birth, while the frontals and sphenoids make their appearance before the third year. Therefore from an anatomic standpoint the sinuses in early life are sufficiently developed to be taken into consideration in pathologic processes.

It is true that they are not always perfectly developed, for even in the adult we know of no structure in the human body which is subject to such wide variations in size, structure, location and development as the accessory sinuses.

In the early years of childhood the upper respiratory tract is particularly susceptible to infection and is generally involved in most of the exanthematous diseases.

In practically every case of measles, whooping cough, diphtheria and scarlet fever, severe reactions are produced in this area. We are always on the alert for ear involvement following these diseases, but do we pay sufficient attention to nasal complications?

The sinuses are lined with mucous membrane similar in structure to that of the eustachian tube, and by the process of continuity can be readily infected.

The numerous recessions and cavities adjoining the nasal fossæ produce an ideal location for the implantation and development of organisms.

If bacteria are placed upon the smooth unobstructed mucous membrane of the mouth we know that they will be eliminated by the normal douching action of the oral secretions as demonstrated by Bloomfield's¹² recent work. However, when they lodge in obstructed crevices like the tonsillar crypts, reduplicated nasal mucous membrane or sinus cavities, they remain for a much longer time.

If the body resistance is below normal, or if the predominating organisms are of a very virulent strain, the defensive action of the tissues may not be able to resist infection. When the sinuses become extensively infected, eradication of the disease is extremely difficult.

Congested nasal mucosa, hypertrophied turbinates, septal deviations, small nasal cavities all interfere with ventilation and drainage, thus prolonging a condition which could be easily eradicated in a more accessible environment.

DIAGNOSIS.

The diagnosis of infected sinuses in children is more difficult than in adults, because subjective symptoms cannot be expressed. In older children, indefinite pains around the head and face with headache may be complained of, although these symptoms are not always present.

The history is very important. It is well to know if the child has had measles, diphtheria, whooping cough and scarlet fever, for White¹³ found thirty-two cases in fifty of his series with infected sinuses who gave a history of childhood diseases. Twenty-nine of these gave a history of whooping cough, three had diphtheria, eight had mumps, while eighteen gave a history of chronic colds.

Onodi¹⁴ tabulated twenty-three cases of the earliest reports of infected sinuses in children and all of these were complications of scarlet fever.

Any chronic cold with nasal discharge should arouse our suspicion of infection in these structures.

Sneezing, headache, irritability and depression are considered very important symptoms by Byfield.¹⁵ Mouth breathing and coughing are also significant signs.

In examination, the nasopharyngoscope is a help, but the procedure is rather difficult. Diagnostic puncture of the antrum with bacteriologic study of the secretion may be resorted to in obscure cases. This procedure has been used successfully by Dean and Armstrong.¹⁶

Transillumination is unreliable, as Skillern¹⁷ points out, and in my own experience has been misleading instead of being of actual diagnostic value.

X-ray examination is our greatest aid, but the plate must be made by a skillful roentgenologist and the interpretation must be correlated with clinical findings.

Any child presenting a majority of the symptoms mentioned or in any case where there is cause to suspect sinus involvement should certainly have the benefit of an X-ray examination.

During the past few years I have had occasion to see a number of cases, young children from three to twelve years of age, who have had their tonsils and adenoids removed without their original symptoms of infection clearing up as had been expected.

They came in for relief of colds, nasal discharge and mouth breathing. Most of them had had their tonsils and adenoids removed from two to three years previous to this examination.

A summary of the important points in the cases presented follows:

REPORT OF CASES.

Case 1.—A. N., age 7, had tonsils and adenoids removed three years ago in order to clear up persistent nasal discharge. As the result of the operation there was some improvement in the symptoms for a while, but the nasal discharge continued. At the present time there is a great deal of thick mucopurulent discharge in each nostril. A quantity of this drains postnasally causing some pharyngeal irritation. The tonsillar fossæ clean and there are no adenoids. Turbinates are congested and enlarged. X-ray examination shows clouding of the antra, also some blurring in the ethmoid cells.

Case 2.—J. M., age 7½, came for the relief of constant nasal discharge. Tonsils and adenoids removed two years ago. At the present time there is no tonsillar tissue remaining, no adenoids found. The infection was confined to the nasal cavity. X-ray examination showed marked clouding of both antra.

Case 3.—E. G., age 7. Tonsils and adenoids removed in 1919. Since operation has had frequent colds and nasal discharge. At the present time there is a great deal of thick mucopurulent postnasal discharge, which produces a great deal of sniffing and coughing. There is no evidence of any remaining tonsil and adenoid tissue. X-ray examination shows definite clouding of both antra and some haziness of ethmoids and frontals.

Case 4.—I. S., age 10, had been treated for chronic rhinitis with constant discharge from right nostril. He gave a history of repeated colds. About two and a half years previous tonsils and adenoids had been removed in an effort to clear up the trouble, but there had been very little relief. Present examination shows a great deal of turgescence of right middle and inferior turbinates. X-ray examination shows clouding of right antrum. After puncturing the antrum and several irrigations the infection cleared up and he has been free of symptoms.

Case 5.—R. L., age 10. Patient was referred for investigation of the sinuses as cause for repeated nasal infections. Two and a half years ago tonsils and adenoids had been removed,

but this had little effect on his nasal infection. X-ray examination shows definite clouding of both antra. There is a great deal of mucopurulent discharge with infiltration of the turbinates and mucous membrane. After a simple puncture of the antra and repeated irrigations the infection cleared up.

Case 6.—R. L., age 6, had tonsils and adenoids removed three and one-half years ago for relief of repeated colds. Since that time there has been very little improvement in the nasal condition. Examination at this time shows marked hypertrophy of turbinates with clouding of both antra. This is confirmed by X-ray examination. No remaining tonsil and adenoid tissue found.

Case 7.—R. S., age 13, was referred for investigation of sinuses as a possible cause for repeated attacks of asthma. Has had these attacks since he was a child. Had resisted all modern methods of treatment. The diet had been carefully investigated, vaccine had been given, tonsils and adenoids removed, but the symptoms would recur. Nasal examination showed a clouding of the left antrum. After puncturing and irrigating this antrum his local nasal symptoms cleared up and he has had no further asthmatic attacks.

Case 8.—C. H., age 6, for the past three years has had intermittent attacks of asthma. Colds have been frequent and there has been considerable nasal discharge. Tonsils and adenoids were removed and an effort was made to clear up the trouble, but there was little improvement in the symptoms. He was referred for nasal treatment. On examination there was marked clouding of left antrum. The right antrum was small and outlines indistinct. Ethmoids were infected and a great deal of enlargement of turbinates.

Case 9.—D. L., age 8, gave a history of repeated colds, a great deal of sneezing and considerable nasal discharge. Tonsils and adenoids had been removed two years previous. Since operation colds have been as frequent, and there has been little improvement in his condition. On examination of the sinuses there was marked clouding of the right antrum. Under local treatment the infection was completely cleared up and the patient has been free from colds for several months.

Case 10.—F. R., age 14, came for relief of repeated attacks of colds. Had tonsils and adenoids removed six years ago. Great deal of mucopurulent discharge in nasopharynx. Examination of nose showed a chronic ethmoid infection and clouding of both antra. Symptoms completely cleared up under local treatment.

TREATMENT.

The simple acute infections of the accessory sinuses in children respond very favorably to treatment. Usually small doses of calomel with a few days' rest in bed is all the general treatment required. Locally, the mucous membrane should be kept clean with some warm cleansing spray of normal saline or dilute Dobell's solution, after which some 20 per cent argyrol should be applied.

The most convenient way to apply this to the nose is to place the child on his back and with an ordinary eyedropper drop from 15 to 20 drops in each nostril. Most of the cases clear up without complications.

In the chronic cases the eradication of the infection is more difficult. If the tonsils and adenoids have not been removed it is advisable to get them out in order to clear up as many foci of infection as possible.

Dean and Armstrong¹⁸ found that 80 per cent of their cases cleared up after the tonsils and adenoids had been removed.

Local treatment consists in keeping the infected surface as clean as possible. Suction is very valuable in this connection, for many crevices can be evacuated which could not be reached otherwise.

Warm saline sprays are very cleansing, soothing and stimulating to the mucous membrane. After this it is helpful to make applications of 2 per cent silver nitrate or 20 per cent argyrol. This should be followed by some emollient spray.

Young children can be best treated in a hospital. The treatment can be carried out much more satisfactorily if the child is away from its parents.

Some cases are helped by the administration of vaccines. A few cases require conservative operative procedures, such as puncture and irrigation of the antra. Only in very exceptional cases is it necessary to do any radical operations.

SUMMARY.

1. All children who have chronic colds with predominant nasal symptoms, such as profuse nasal discharge, stuffiness of the head, mouth breathing and asthmatic attacks, should have a careful investigation of the sinuses.

2. Cases in which tonsils and adenoids have been removed for focal areas of infection, and the symptoms have not cleared up after operation, infection of the accessory sinuses should be suspected.

3. Neglected treatment of early infection of the sinuses in children predisposes to chronic infection which may lay the foundation for serious complications in later life.

4. Nasal colds in children should be treated as an important disease. Careful investigation should be made as to their etiology and diagnosis.

5. The proper treatment of infected sinuses in children gives gratifying results. The general health of the patient is improved. They take on weight, develop more rapidly and give every appearance of being greatly benefited. Most of the cases can be cured by local treatment, for only a small number require operative procedures.

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XXXII.

SOME REMARKS ON NYSTAGMUS.*

By G. W. MACKENZIE, M. D.,

PHILADELPHIA.

When Dr. Skillern, the spokesman for the Program Committee, invited the writer to present a paper before the academy it was suggested that the subject be one the nature of which should appeal to both eye and ear specialists. A further suggestion of the committee was to the effect that a highly technical paper, delving into speculative and fanciful theories, would be less acceptable to the average fellow in attendance than one which adheres more closely to fundamental and established facts. These suggestions were agreeable to the writer. Accordingly, a paper on nystagmus was prepared in which the attempt is made to present the subject in the simplest manner possible, based mostly upon personal observations of a large number of normal individuals, and of others with definitely known pathologic conditions, covering a period of eighteen years.

We can best describe nystagmus as involuntary to and fro movement of the eyes. According to the comparative rapidity of the two opposite movements (to and fro), we may conveniently divide nystagmus into two general forms, the undulatory and the rhythmic. In the undulatory, sometimes called oscillatory, form of nystagmus the to and fro movements occur with equal rapidity, resembling therefore the pendulum movements of a clock. Undulatory nystagmus is most commonly found in individuals suffering from impairment of vision, more especially central vision. The earlier in life the impairment of vision occurs the more prone the individual is to develop nystagmus. The lesion responsible for the visual defect may have occurred during intrauterine life.

*Read before the Academy of Ophthalmology and Otolaryngology, Minneapolis, Sept. 19th, 1922.

The location of the lesion may be in the macula, the maculopapular bundle, or in the ultimate center for central vision.

It is claimed that central opacities of the optical media alone may produce nystagmus. This claim has not been entirely confirmed in the experience of the writer. In the vast majority of cases of corneal opacity we find no evidence of nystagmus. Theoretically the location of the opacity ought to exert some determining influence. For instance, we should be able to find more cases of undulatory nystagmus among those individuals with central than in those with excentral opacities. However, this is not borne out by experience, for we find in the vast majority of cases of central scars of the cornea, even the small dense ones, no sign of nystagmus. The same holds true of anterior and posterior polar cataracts. The size of the opacity appears to exert little influence, for we find quite as many cases of nystagmus among individuals with large as among those with small scars. In spite of this, there remain two apparently inconsistent facts. The first is that undulatory nystagmus is found more commonly among those individuals affected with corneal opacities, together with poor vision, than in those free of these defects. The second is that only a relatively small per cent of the total number of individuals so affected manifest nystagmus. How can these two facts be harmonized and at the same time prove that opacity of the optical media is an actual cause of nystagmus? The answer is that it cannot be done. We must look elsewhere for the cause. The opacity of the optical media is a mere incident and not the cause of the nystagmus. Let us consider the following facts and draw our conclusions accordingly:

(a) The most classical cases of undulatory nystagmus are to be found in those conditions where there is a relative absence through degeneration or otherwise of the retinal pigment in the macular region; for instance, in albinos, coal miners, and cases of central choroiditis.

(b) In cases of diseases of the anterior part of the eyeball, the posterior part is more often diseased than in those cases where the anterior part of the eyeball has remained healthy.

(c) In cases of marked undulatory nystagmus it is decidedly more difficult to make a detailed examination of the eye grounds than in an eyeball that remains stationary.

(d) In those eyes with opacities of the optical media it is more difficult to see the finer details of the fundus than in eyes presenting normally transparent media.

There is but one logical conclusion, and that is, the uveal tract is more often involved in cases of opacity of the cornea than is demonstrable by ophthalmoscopic examination, and undulatory nystagmus is due more to the poor central vision from lack of function of the macula than from the impairment of vision from corneal opacity alone. In other words, the capacity of the cornea is a mere incident and not the sole cause of the nystagmus. It is possible, too, in individuals blinded by any disease of the eye, anterior to the retina, that the lessened amount of illumination reaching the retina causes a relative loss of pigmentation of the retinal epithelium.

Undulatory nystagmus is induced by the futile efforts of the patient to find that sharpest vision, which is possible in normal individuals only when the image of an external object falls upon the macula, which is impossible in the case of one whose macula is for any reason more or less functionless. The failure to accomplish the impossible in spite of repeated efforts to do so over a prolonged period must necessarily result in the development of habitual movements of the eyes in the form of undulatory nystagmus.

Summarizing: Undulatory nystagmus is an involuntary to and fro movement of the eyes. This to and fro movement occurs with equal rapidity. It results from visual defects, especially central. The lesion responsible for the visual defect occurs in early life or even during intrauterine life.

Rhythmic nystagmus, like the undulatory form, is involuntary to and fro movement of the eyes, but unlike the undulatory, and to and fro movement occurs with unequal rapidity; in other words, the movement in one direction is comparatively slow while the return movement is rapid.

In recent years, rhythmic nystagmus has been the subject of considerable study because of its common occurrence in diseases of the so-called vestibular apparatus and its nervous pathways.

Although the writer has described both forms of nystagmus in a broad way as involuntary to and fro movement of the eyes, he wishes to qualify somewhat his earlier statement in

so far as undulatory nystagmus is concerned. Every ophthalmologist can recall the difficulty he has experienced at times in studying the ocular fundi of a patient affected with undulatory nystagmus; furthermore, he can recall how at a properly directed command the patient is able to inhibit somewhat the intensity of the ocular movements for a brief period only (several seconds), when the intensity of the movements again increases up to that which is usual for the particular patient under observation. Thus we see that undulatory nystagmus, although it is involuntary, can be more or less controlled by a strong inhibitory effort on the part of the patient. The ability of the patient to inhibit the intensity of his eye movements in the case of undulatory nystagmus is comparable with the ability of a patient to inhibit the pillrolling movements of the fingers in the case of paralysis agitans. Rhythmic nystagmus, on the contrary, cannot be inhibited by the patient, no matter how strongly he may will to do so.

The eye movements in undulatory nystagmus may occur in any plane—the frontal (torsional), sagittal (vertical), or the horizontal. Besides, these movements may occur in a combination of planes—torsional with horizontal is perhaps the most common of all. Since, in the undulatory form of nystagmus, the to and fro movements occur with equal rapidity, we cannot consistently refer to the direction of the nystagmus; for instance, to the right or to the left, as we can in the case of rhythmic nystagmus.

Rhythmic nystagmus, no matter from what cause, remains true to form under all circumstances. This is not true of the undulatory form, for we find that it is changed to the rhythmic form when the patient attempts in the slightest degree to look in any other direction than straight ahead; especially is this the case if he looks laterally to either side. For instance, if a patient with undulatory nystagmus, the result of a central visual defect, attempts to look to the right, he will manifest a rhythmic nystagmus to the right, which just as readily changes to a rhythmic nystagmus to the left, when he attempts to look to the left. The question naturally arises, why is this so? It is so, for the reason that most, if not all, normal individuals manifest a rhythmic nystagmus to the right, when they look to the extreme right, and a rhythmic nystagmus to the

left, when they look to the extreme left. This rhythmic nystagmus, which is evident in normal individuals, may be horizontal, or mixed horizontal and rotary, or rotary, depending upon how far laterally he directs his vision. It is purely physiologic so long as the intensity of the nystagmus is equal to the two sides for the same degree of angle deviation of the eyes, and disappears when the patient looks straight ahead at infinity.

This physiologic nystagmus exerts a modifying influence in every case of spontaneous nystagmus, undulatory or rhythmic; for instance, we have observed above that the influence of the physiologic element is sufficient to change the character of nystagmus from undulatory to the rhythmic form, and this too by only a moderate effort at side glancing. Again, in the case of spontaneous rhythmic nystagmus, let us say to the right, the slightest attempt to move the eyes to the right is sufficient to increase perceptibly the intensity of the nystagmus to the right, while the slightest attempt to move the eyes to the left will decrease the intensity of the nystagmus to the right. The nystagmus is increased to the right in the case just cited, because there are two influences cooperating to produce nystagmus to the right: the original one that produced the spontaneous nystagmus to the right, plus the physiologic nystagmus or the tendency to the physiologic nystagmus to the right when the patient looks to the right. Rhythmic nystagmus to the right is decreased when the patient looks to the left, because there are two influences operating against one another; the original one that produced the spontaneous nystagmus to the right and the inhibiting influence of the physiologic nystagmus to the left. In some cases, the physiologic influence toward the left when looking to the left is sufficient to neutralize the pathologically induced spontaneous nystagmus to the right, thereby arresting it. This would happen in the case where the pathologically induced spontaneous nystagmus to the right is less pronounced than in the preceding case. In still other cases the influence of the physiologic element toward the left may be sufficient to overbalance the pathologic element toward the right, the result of which is a rhythmic nystagmus to the left, so long as the patient looks intently in that direction. In this case, the pathologically pro-

duced spontaneous nystagmus to the right is minimal in amount but of clinical significance.

Rhythmic nystagmus, like undulatory nystagmus, may occur in any plane or combination of planes, but unlike the undulatory nystagmus, it occurs in a definite direction and is generally designated according to the direction of its quicker component. Thus we may speak consistently of horizontal nystagmus to the left, rotary to the right, etc. To designate it less fully would be insufficient for accuracy. The moment we designate its direction, we at the same time stamp the nystagmus as rhythmic in form, for the simple reason that it is not possible to conceive of any definite direction for undulatory nystagmus, where the two movements occur with equal rapidity. With some beginners there seems to be confusion as to how we should designate the direction of rhythmic nystagmus, due no doubt to the fact as established by Barany that the slow movement of the eyes is the actual vestibular reflex, while the quicker movement is purely voluntary; for instance, if the right labyrinth and nerve are stimulated with the negative pole of the galvanic current, there follows directly a slow movement of the eyes to the left side. So long as the individual being tested is conscious, he experiences a visual sensation of external objects moving slowly to the right. To reorientate himself, he quickly brings his eyes to the right by a voluntary effort originating from an impulse in the cerebral cortex. The continued application of the negative current stimulates the right vestibular nerve; then Deiter's nucleus sends the impulse along the posterior longitudinal bundle to the eye muscle nuclei, causing the eyes again to move slowly to the left, when the brain recognizes the outside world sliding to the right; by an effort of the will, the subject under examination brings his eyes back to their primary position. In short, the slow movement is the actual vestibular reflex and is, therefore, involuntary; while the quick movement is purely voluntary. In designating the direction of rhythmic nystagmus, it is customary to designate it after the direction of the quicker movement, and not according to the direction of the actual reflex movement, as some might prefer.

Rhythmic nystagmus can be produced experimentally in several different ways: (1) By directing the normal indi-

vidual to look to the extreme right or extreme left, referred to earlier and termed physiologic. (2) By placing before the eye a prism base in or out of sufficient strength to throw a strain on one or the other of the horizontally acting eye muscles, when the nystagmus will occur in the plane corresponding to the action of the muscles upon which the strain is placed, the direction of the nystagmus (quicker component) will correspond to the normal action of the same muscle (i. e., toward the apex of the prism). In only a few experiments of this kind are we able to produce a typical rhythmic nystagmus, but in a sufficient number of cases to satisfy the principle. (3) By stimulating the vestibular apparatus and nerve with negative galvanism or by inhibiting the normal tonus with positive galvanism. (4) By producing endolymph impact or movement against the hair cells of the crista ampularis of the semicircular canals, either by the so-called caloric or turning tests.

Furthermore, rhythmic nystagmus may be produced experimentally in a pathologic case; for instance, in a patient afflicted with undulatory nystagmus of ocular origin, the undulatory character of the nystagmus may be changed to the rhythmic form by having the patient look in any other direction than straight ahead, which has already been alluded to.

Rhythmic nystagmus, pure and simple, may result from certain well known pathologic conditions. Rhythmic nystagmus may occur from paresis of any of the extraocular muscles, when it may be accompanied with vertigo. The nystagmus will take place in the same plane and the same direction as the normal action of the weak muscle. The nystagmus reaches its maximum intensity with the maximum effort to use the paretic muscle. Rhythmic nystagmus is never present in such a case when the patient looks in the opposite direction, that is, away from the paretic muscle, and is rarely, if ever, evident when the patient looks straight ahead, which is contrary to that which occurs in the next class of cases.

Rhythmic nystagmus results from pathologic conditions affecting the inner ear or eighth nerve. The character of these conditions is not always the same. They may be conveniently divided into two classes, the irritative and destructive. Again, conditions of a similar kind may differ in degree. For in-

stance, we find irritative conditions in the inner ear, which so far as their effects are concerned, compare with those of stimulation with the cathode. Both tend to produce a rhythmic nystagmus toward the side of irritation or stimulation. The intensity of the resulting nystagmus depends upon the intensity of pathologic irritation or cathodal stimulation. In both instances the resulting nystagmus is not purely horizontal, for the vertical canals are stimulated as well as the horizontal, nor is the nystagmus purely rotary, for the horizontal canal is included with the verticals in the stimulation. The resulting nystagmus from stimulation of all these canals ought to occur in both the horizontal and frontal planes, and in fact it does. In other words, there is a mixed horizontal and rotary nystagmus. If the pathologic irritation is limited to a single canal, we ought to get a single plane nystagmus, and in fact we do, observable in cases of irritative lesions (congestion) of the external semicircular canal, in the early stages of labyrinthine fistula.

In a diffuse destructive lesion of the inner ear, so long as it is of recent origin, there results a rhythmic nystagmus, mixed horizontal and rotary, away from the affected side, comparable with that produced by positive galvanism. In old standing cases (a year or more following the destruction) the spontaneous rhythmic nystagmus that existed early will have disappeared entirely or nearly so. In the circumscribed destructive lesions of recent origin there occurs a spontaneous rhythmic nystagmus away from the side of the lesion, in the plane corresponding to the plane of the canal affected.

Lesions of the vestibular branch of the eighth nerve produce mixed horizontal and rotary nystagmus toward the side of the affection in cases of mild congestion, and away from the side of the lesion in those cases of inflammation sufficiently pronounced as to cause diminution or loss of function. The time limit will not permit me to indulge in this interesting phase of the subject further at this time.

Recently there has been some doubt cast upon the possibility of nystagmus resulting from lesions of the cerebellum. Notwithstanding, I am convinced that nystagmus can occur from a lesion of the cerebellum pure and simple. My reasons are many, but I will content myself with the citation of a

single case at this time. In consultation with Dr. W. H. Sears of Huntingdon, Pa., several years ago, I operated upon a case of left temporosphenoidal lobe abscess, complicated with a left sided labyrinthine suppuration. Dr. Sears, after a most searching examination, diagnosed correctly the labyrinthine suppuration and felt sure as to the presence of an abscess somewhere in the brain, but was inclined to believe the abscess was in the cerebellum.

As a result of the right sided labyrinthine suppuration, the patient manifested before the operation a spontaneous mixed rotary and horizontal nystagmus to the opposite (left) side. At the operation the labyrinth was exenterated and nothing at all was noticed about the behavior of the eyes, the patient being under light narcosis. The cerebellum was then incised in a search for the abscess, when immediately the patient sprung a horizontal nystagmus to the right (operated) side of exceptionally wide excursions. A few minutes later the temporosphenoidal lobe was incised and a large quantity of foul smelling pus was emptied from the abscess cavity, which produced no further effect upon the nystagmus. In brief, the right sided labyrinthine suppuration had caused, as it always does, a rhythmic mixed horizontal and rotary nystagmus toward the opposite (left) side; exenteration of the labyrinth produced no change in the existing nystagmus. Incision of the right cerebellar hemisphere immediately changed the plane and direction of the nystagmus from a mixed horizontal and rotary toward the left to a purely horizontal toward the right. Further operative work in the middle fossa did not influence the character of the nystagmus produced shortly before, by incision of the cerebellum. The irritation produced in the cerebellum by the incision was the clearest evidence to me and those present that a lesion of the cerebellum alone will produce nystagmus; others have observed it, too.

Speaking of nystagmus of cerebellar origin, the writer has observed it to be more frequently purely horizontal, less frequently purely vertical, and least frequently mixed horizontal and rotary. Purely horizontal nystagmus, when of cerebellar origin, points toward a lesion of one of the hemispheres. Since abscess of the cerebellum occurs more frequently in one of the hemispheres than in the middle lobe or vermis, we nat-

usually find horizontal nystagmus to be the prevailing form. In the earliest stages—that is, the period of congestion, when we have signs of irritation, the stimulating effects of the irritation produce horizontal nystagmus toward the side of the lesion: very soon the effects of destruction are felt, when the nystagmus swings over toward the opposite side (away from the side of the lesion); however, so long as the process is limited to one hemisphere the nystagmus retains its horizontal character. In diseases of the vermis alone, for instance, a small circumscribed tumor, the nystagmus is purely vertical. Since abscess of the cerebellum is rarely if ever limited to the vermis, a vertical nystagmus associated with increased intracranial pressure would point toward a tumor rather than an abscess. Rarely does one find a mixed rotary and horizontal nystagmus in abscess or tumor of the cerebellum; when it does occur, it is not due to the lesion in the cerebellum proper but to pressure on the root of the vestibular branch of the eighth nerve or its nuclei; in other words, it is a more or less distant working symptom, in the sense that MacEwen first intended its interpretation.

For the benefit of the eye men, the writer feels that he would like to call attention to the fact that nystagmus can be readily observed during the making of an ophthalmoscopic examination, especially by the direct method. Every now and then one may observe rhythmic nystagmus of the eye at the posterior pole, where its direction is the reverse of what it is at the anterior pole. In all such cases a note should be made of the fact and the case studied more closely with the object of determining its cause.

With one exception I have deliberately avoided the citation of cases, as interesting as they are. My intention has been to carry out as far as possible the wishes of the Program Committee, who feel, as the writer does, that the greatest need is to the greatest number—that it is better to present a simple paper, at the risk of making it kindergarten in style, rather than attempt a more technical one, that might appeal to but a limited few.

1724 SPRUCE ST.

XXXIII.

THE DIAGNOSIS AND TREATMENT OF PULMONARY CONDITIONS THROUGH THE BRONCHOSCOPE.

FROM THE MEDICAL SCHOOL, CREIGHTON UNIVERSITY,
DEPARTMENT OF OTOLARYNGOLOGY.

By B. M. KULLY, M. D.,

OMAHA.

The direct examination of the upper air passages has now been in use a number of years. The direct examination of the larynx was first introduced by Kirstein in 1894. Tracheoscopy, described by Kirstein and Mikulicz, soon followed. In 1897 Killian demonstrated the feasibility of upper bronchoscopy and removed a foreign body from a bronchus. Killian later developed lower bronchoscopy, his achievements earning for himself the title of the "Father of Bronchoscopy." In 1899 Coolidge removed the fragment of a tracheotomy tube from a bronchus. Von Schrötter and Piniasek published their work in 1901, and in 1904 Chevalier Jackson introduced his instruments and initiated his career. Briefly, these are the noteworthy contributions in the early development of bronchoscopy.

The value and use of the bronchoscope in the extraction of foreign bodies from the upper air passages were generally and immediately recognized. Reports of successful removals soon became numerous. The progress and achievements in this particular field are well known and need no discussion. The work of Killian and Bruening in Germany, of Jackson, Lynch, Yankauer and a host of others in this country forms one of the fascinating chapters of medical history.

The bronchoscope, however, has not been generally recognized nor adopted as an important factor in the field of pulmonary diagnosis and therapy. And this, notwithstanding the fact that its use as such is almost as old as is the instrument itself. As early as 1900, Killian¹ presented a paper on the

diagnosis of lung carcinomata with upper bronchoscopy. In 1905 Newmayer reported a bronchoscopy in a case of epithelioma of the lung. In 1906, M. Kob bronchosoped a case of lung echinococcus. Jackson in 1907 reported an ulceration at the tracheal bifurcation, diagnosed and treated through the tracheoscope. In spite of this pioneer work, development of this phase has been slow. To most of the profession mention of the bronchoscope has always been associated with foreign body work only. It is only in recent years that we have commenced to appreciate the bronchoscope as an asset in the field of pulmonary diagnosis and therapy. The laryngologists as well as the general profession are not sufficiently cognizant of this. As upon the laryngologists must devolve not only the use of this instrument, but the education of the medical profession as to its value, a brief resumé of the work in this field is timely.

Without the aid of the bronchoscope a host of conditions must remain either unrecognized or untreated. Benign and malignant neoplasms and inflammatory processes of various sorts are included in this category. Compression stenosis of the trachea and bronchi indicates bronchoscopic examination as an aid to a proper conception of the condition and a rational method of treatment.

BENIGN NEOPLASMS.

Benign neoplasms, particularly, give rise to a varied clinical picture, for the interpretation of which our present clinical and Roentgen findings are frequently inadequate. Benign tumors of the tracheobronchial tree are rarely discovered except at autopsy. That such conditions can be recognized and treated through the bronchoscope is evidenced by the increasing number of case reports. Tilley³ has reported the diagnosis and removal of an intratracheal tumor by peroral endoscopy. Horne⁴ has reported a papilloma of the bifurcation of the trachea. The writer has seen a pedunculated fibroma completely filling the left main bronchus. This patient presented the classical symptoms of empyema and was repeatedly aspirated for such with negative results. The tumor was removed endoscopically by S. Yankauer, and after a few lung irrigations the patient made a complete recovery.

MALIGNANT NEOPLASMS.

Malignant neoplasms offer another important field for the bronchoscope. Diagnosis by other means is frequently mistaken or delayed. Funk⁵ calls attention to the fact that malignant disease is often mistaken for tuberculosis. It has been our experience that it is often associated with and masked by chronic pulmonary suppuration. In a series of ninety-two cases of pulmonary suppuration bronchoscoped at Mt. Sinai Hospital, New York, there were discovered six previously unsuspected malignant neoplasms. If the growth originates in or has invaded the bronchus it can be recognized through the bronchoscope, and it is usually possible to remove a section for microscopic examination. The treatment of malignancy of the lung by any method is, of course, unsatisfactory. Radium therapy can be made more effective, however, by introducing the radium capsule directly into the growth through the bronchoscope.

CHRONIC PULMONARY SUPPURATION.

In recent years the direct examination has been increasingly used in a number of inflammatory conditions. A great deal of work has been done, particularly in the treatment of chronic pulmonary suppuration. Chronic pulmonary suppuration, including lung abscess, bronchiectasis and gangrene, has long been one of medicine's failures. With few isolated exceptions, medical and surgical treatment has been of no avail. Bronchoscopy offers a direct and logical method for coping with the condition. The method as first introduced by Yankauer in 1916 is as follows: The bronchoscope is introduced into the suppurative focus or into the bronchus, draining it, and by means of a double current canula fluid is injected and aspirated. Continuous irrigation and aspiration are maintained until the solution returns clear. If more than one lobe is involved, the process is repeated in each involved lobe. The irrigating fluid may be normal saline or one containing an antiseptic. Weak iodine solutions have usually been used, but at present we are using a solution of potassium permanganate. In addition to the irrigation, all conditions preventing adequate drainage are treated. Cicatricial stenoses of the draining

bronchi are dilated. Granulation tissue is cauterized or removed.

The procedure is not a very trying one. The patients report to the hospital, are bronchoscoped and irrigated and are discharged the same day. They are able to resume their work the following day.

Considering the almost hopeless pathologic condition, the results have been excellent. The profuse, purulent expectoration and the fetid odor can usually be controlled. In a series of twenty-nine cases which the writer⁶ reported last year, there were four clinical cures, nineteen others were sufficiently improved to be able to resume their respective occupations and places in society. Some of the patients received over one hundred and fifty bronchoscopic irrigations without a single severe reaction in the entire series.

OTHER INFLAMMATORY CONDITIONS.

Syphilis of the lung does not ordinarily require endoscopic interference. It is interesting to note, however, that as early as 1905 A. Reinhard⁷ published the bronchoscopic findings in a case of syphilis of the lower air passages. Luetic cicatrices of the trachea and bronchi do occur and may urgently require direct treatment. The writer has repeatedly dilated a luetic stricture of the trachea that was causing intense dyspnea.

Investigations of the trachea and bronchi have been made in diphtheritic and postdiphtheritic conditions. Lynch has reported and treated pulmonary suppuration in association with postdiphtheritic bronchial stenosis. Recently Purcell and Acree⁸ have reported a case of diphtheritic tracheitis and bronchitis which required repeated bronchoscopic removal of the membrane for the relief of dyspnea.

Simple ulcers of the trachea and bronchi as reported by Jackson in 1907 have repeatedly been found and treated by direct application. Herein may lie the explanation and the treatment for some of our inexplicable chronic coughs.

An attempt has also been made to treat asthma by direct application through the tracheoscope, and a few successful results have been reported. Knowing the uncertainty and vagaries of the disease and its tendency to disappear at times, the results are far from conclusive.

TRACHEOBRONCHIAL COMPRESSION.

Tracheobronchial stenosis from external compression indicates direct examination. Important information as to diagnosis and indications for treatment is frequently obtained. Frazer⁹ calls attention to the importance of the position of the trachea in intrathoracic disease. In a case of thoracic dermoid operated upon and recently reported by Lilienthal, the bronchoscopic finding was an important factor in establishing the preoperative diagnosis. As dermoid cysts are situated in the anterior mediastinum the tracheal compression was from in front. Congenital stridor in infants is usually due to a compression of the trachea by an enlarged thymus, but may be due to laryngeal conditions, such as papillomata, angiomata, etc. The diagnosis can be made by direct examination. The bronchoscope has at times proved an invaluable aid in the diagnosis of compression due to aneurysm, malignancy of the mediastinum and of the esophagus and interlobar empyemata.

INSUFFLATION OF OPAQUE SUBSTANCES AS AN AID TO ROENTGEN DIAGNOSIS.

Investigation of the tracheobronchial tree with the X-ray after the injection of opaque materials is still in the experimental stage. Considerable evidence has been adduced, however. Jackson¹⁰ reports that insufflation of dry bismuth subcarbonate produced no symptoms, mapped out the bronchial tree completely and disappeared in twenty-four hours. Bullowa and Gottlieb,¹¹ working with dogs, have also reported a rapid disappearance of the opaque material and believe that the bronchial musculature has a peristaltic action. It is possible by this method to map out lung abscess and bronchiectatic cavities. The Roentgen investigation of the lungs by this method is still in the embryonic stage. It may or may not develop into a valuable asset in the field of pulmonary diagnosis.

CONCLUSION.

The bronchoscopic investigation and treatment of the conditions herein mentioned is of course valuable and in many instances essential. More important, however, is the realization of the field which is opened up and the possibilities it

holds. The indications for its use should be remembered. Every case presenting persistent obscure lung signs warrants bronchoscopic investigation. The method is still in its incipency. We believe that it is destined to expand into one of vast usefulness. Sufficient has already been accomplished, however, to have proved it invaluable in the handling of hitherto unrecognized and untreated pulmonary conditions.

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XXXIV.

THE STRUCTURE AND FUNCTION OF THE CRISTA AMPULARIS.*

BY GEORGE E. SHAMBAUGH, M. D.,

CHICAGO.

Because the reactions in the labyrinth which result in a stimulation of the end organs are physical reactions, it is obvious that any effort to analyze these reactions must be based on as accurate a knowledge of the structures of these end organs as it is possible to gain. Such a knowledge can be acquired only by the study of a series of actual preparations. It cannot be gathered from descriptions or from illustrations.

To one who has acquired from the study of actual preparations an appreciation of the complicated, delicate mechanism in the membranous labyrinth, certain facts become at once apparent. One is that conclusions drawn from the making of models are of very limited value, for the reason that it is quite impossible to construct models which have any but the remotest resemblance to the delicate, complicated structure in the membranous labyrinth. Another is that experiments based on the results of operation on the several parts of the internal ear of lower animals are of but relatively little assistance in our attempt to analyze the normal reactions resulting in a stimulation of these end organs, for the obvious reason that no operation of this sort can be carried out without causing the escape of the labyrinth fluids and thus necessarily putting an end at once to any and all of those delicate reactions which bring about the normal stimulation. The final conclusion forced upon one who has acquired an appreciation of the delicate structures in the labyrinth is that our main reliance for an explanation of the reactions in the labyrinth resulting in a stimulation of its end organs rests upon experiments on the human. We are fortunate in being able to carry out a large

*Presented before the Chicago Laryngological and Otological Society, January 8, 1923.

series of experiments without in any way harming the individual.

In our attempt to analyze the reactions in the internal ear it is highly important that we avoid as far as possible any intricate explanations and keep the whole discussion as simple as possible, for it is only in this way that progress is made. One thing is certain, there is no point to be raised in this discussion that does not open the door for endless arguments. The conclusion that approaches more nearly the correct explanation is the one which accounts readily for most of the phenomena.

Then followed a lantern demonstration of the minute anatomy of the end organs in the semicircular canals, and the reason was pointed out for concluding that the cupola constituted a fixed cap on the crista not capable of displacement, which means that the interaction between the cupola and the hair cells of the crista continues only so long as the endolymph current lasts and no longer.

The discussion began with the phenomena resulting from the rotation experiment. These phenomena of rotation may be stated briefly as follows: On starting rotation, say to the right, the individual in the upright position experiences the sensation of being rotated to the right, and a horizontal nystagmus with the quick component to the right develops; both of these persist for a few seconds and then disappear and remain absent, no matter how long the turning is kept up, provided there is no acceleration or retardation in the speed of rotation. On stopping the rotation the individual experiences the sensation of again being rotated, but this time to the left, and there develops a nystagmus towards the left. These phenomena persist for perhaps 20 seconds before they die out. That these phenomena are the result of the movement of the endolymph in the horizontal canals is questioned by no one. Three explanations of the reactions producing these phenomena are offered.

Breuer believes that but a momentary impulse to the endolymph can result from inertia of this fluid, both on starting and on stopping rotation, before this motion is overcome by the friction of the fluid on the walls of the very minute membranous canal. He accounts for the persistence of the phe-

nomena by assuming that this momentary impulse impinging on the cupola displaces this structure on the crista. The resulting stimulation of the hair cells and the phenomena resulting from this stimulation persists until the cupola has been drawn back to its normal position. Barany also accepts but a momentary impulse to the endolymph, but accounts for the persistence of the phenomena on the assumption that this momentary impulse on starting and stopping rotation explodes so-called nystagmus centers, one producing nystagmus towards one side, the other to the opposite side. These centers contain stored energy which requires just so long to expend itself. The other explanation is that the inertia of the endolymph persists both on starting and stopping rotation for some seconds, and the duration of the stimulation of the hair cells is the same as the duration of the endolymph current and equals the duration of the phenomena resulting from starting and stopping rotation.

The three hypotheses may be expressed graphically as follows:

1. Duration of endolymph current $<$ duration of peripheral stimulation = duration of nystagmus (Breuer).

2. Duration of endolymph current = duration of peripheral stimulation $<$ duration of nystagmus (Barany).

3. Duration of endolymph current = duration of peripheral stimulation = duration of nystagmus.

Of these three hypotheses the only one that offers a plausible explanation of the phenomena, not only of the rotation experiment but of the other experiments to be discussed later, namely, the Ewald experiment, the caloric experiment and fistula cases, as well as the explanation of the tonus itself, is the one which makes the duration of the endolymph current, the peripheral stimulation and the nystagmus all equal. How this is so will be pointed out when discussing the several experiments.

The question at once arises about the objection to the theory of endolymph currents on account of the minute caliber of the membranous canals. This objection has become formidable chiefly because of the failure to grasp fully the several forces taking part in the movement of endolymph in the canals resulting from inertia. These objections have been based on the

minute caliber of the membranous tubes, as though the caliber of these tubes was the only factor to be considered. As a matter of fact, it seems extremely probable that the inertia of the endolymph in the membranous canals has little, if any, bearing on the duration of the endolymph in the canals resulting from the rotation experiment. The main factor in continuing the endolymph current is the inertia transmitted to the fluids of the much larger channels of the osseous canals and particularly to the fluids in the vestibule itself. These fluids are as certainly affected by the rotation as is the endolymph in the membranous canals, and the movement of the fluid in these much more roomy spaces by pressure on the membranous structures forces the endolymph to continue moving. With this fact in mind it seems much easier to accept the last formula, which alone explains all the phenomena, than either of the other hypotheses which, as we shall show, offer no plausible explanation for much of the phenomena.

Ewald's experiment, which produced a measured movement in each direction for the several canals, brought out several facts of fundamental importance in the physiology of these structures. The first is that an endolymph current in a particular canal produced forced movements, head motion, nystagmus, only in the plane of that canal. The second is that the movement of the head or the slow motion of the nystagmus is always in the direction from right to left, or from left to right of the endolymph current. A third fact brought out by his experiment was that in each canal a stronger response was elicited by an endolymph current in one direction from that caused by a current in the opposite direction and that the greater response in each canal was always from those currents which produced movements toward the opposite side. This last fact furnishes the explanation why the tonus from the right ear when not checked by the tonus from the other side always produces movements to the left and vice versa. This will be brought out more clearly in the discussion of tonus.

The Fistula Phenomena.—In chronic suppurative otitis media it is not uncommon for an erosion to take place through the bony capsule of the labyrinth, usually the horizontal canal. In such cases it is possible by compression and rarefaction of air in the external meatus to reproduce the Ewald experi-

ment. In both the Ewald experiment and in fistula cases it has been found that the duration of the phenomena resulting from the movement of the endolymph is exactly the same as is the endolymph movement. Some years ago I saw a patient where on one side there was a dead labyrinth, the result of an old suppurative labyrinthitis, and in the remaining ear there was a fistula in the horizontal canal. In this ear there was fortunately a complete closure of the eustachian tube. The situation was particularly suited for regulating the compression of air in the external meatus and for observing the resulting phenomena without any question of interference from the tonus from the opposite ear. I examined this case a great many times, using a bulb which completely closed the external meatus and a suitable cut off from a compressed air apparatus: I found that the nystagmus resulting from compression always lasted exactly as long as did the compression. In this case with the air pressure employed, it always took about ten seconds for the complete collapse of the membranous canal, for at that moment the nystagmus always stopped. Now when the compression was kept up a shorter time and then held, at that moment the nystagmus also ceased. The only explanation for this phenomenon is that the stimulation of the hair cells continues only so long as the actual movement of the endolymph lasts, and the resulting nystagmus continues only so long as the peripheral stimulation continues. With either the Breuer or the Barany hypothesis we should expect the nystagmus to continue for a definite time after the cessation of the compression—that is, after the cessation of the endolymph current. In the first instance, because of the displacement in the cupola, and in the second, because of the expenditure of stored energy in nystagmus centers.

The Caloric Experiment.—Barany found that by chilling or warming the outer wall of the labyrinth, endolymph could be made to flow in either direction in the two canals which lie close to the middle ear chambers, the superior and the horizontal canals. In the upright position the superior canal lies in the vertical plane and permits of endolymph currents from caloric stimulation, while the horizontal canal, lying on the horizontal plane, no such movements are possible. On tipping the head either forward or backward, the horizontal canal

comes in the perpendicular plane and the superior lies horizontally. Now it is in the horizontal canal in which endolymph current is possible from caloric stimulation. As long as the superior canal alone was perpendicular a purely rotating nystagmus developed, and when the horizontal canal alone was perpendicular a purely horizontal nystagmus results. When the position of the head was such that both canals were partially in the perpendicular plane, a mixed rotating and horizontal nystagmus results. These are exactly the results we are prepared to expect from the facts brought out in the Ewald experiments. Now the interesting fact is this, that the nystagmus produced by the head in the upright position stops the moment when this canal is placed in the horizontal plane, that is the exact moment the endolymph current ceases. With both the Breuer or the Barany hypotheses this could not take place.

In all of these experiments there is but one explanation possible—that is, that the duration of the stimulation and the resulting nystagmus equals the duration of the endolymph current. Confronted by this accumulated evidence regarding the existence and duration of endolymph currents, I find myself unable to take seriously the theoretical objections* based upon the opinion of physicists who have considered only the caliber of the semicircular canals.

Labyrinth Tonus.—If I were asked what in view of our clinical experiences I would consider as the important role of the vestibular mechanism, I would unhesitatingly say a mechanism of equilibration, which is accomplished by virtue of tonus impulses which are constantly emanating from the labyrinth to supply the skeletal muscles. It seems quite probable that the end organs in the vestibule, the macula acustica, take part in supplying this tonus, but there is every reason for believing that the crista ampularis plays the more important role in producing this tonus.

Several explanations have been offered for the origin of labyrinth tonus. Ewald assumed that the hair cells of the crista were in a state of ciliary motion, which constituted a constant stimulation of the hair cells. This is of course impossible, since these hairs do not project free in the endolymph but are covered by the cupola. Breuer assumed the existence of endo-

lymph currents from the stria vascularis where it is secreted out through the aqueductus vestibuli to the cerebrospinal fluid. This hypothesis is also impossible on anatomic grounds. Barany placed this tonus in tonus centers which was replenished from the labyrinth, but he did not explain how. Now it has been apparent to all of these men that all that is required to account for the flow of tonus from the labyrinth is a constant, however mild, stimulation of the hair cells of the cristæ. Now the normal stimulation of the crista is the result of endolymph movements against the sides of the cupola. It seemed to me quite probable that this motion of the endolymph could be the result of the to and fro movements imparted to the fluid from the pulsation in the labyrinth artery. I advanced this explanation when I first published my conclusion regarding the function of the semicircular canals in 1912. Clinical evidence all points to this as the correct explanation of the origin of labyrinth tonus. I shall not enter into this now, suffice it to say that Barany now accepts this explanation and has published clinical observations justifying this hypothesis. These pulsations in the endolymph stimulate equally the hair cells on both sides of each crista. We have seen, however, that the greater response in each canal results from the stimulation of those endolymph currents which produce movements towards the opposite side. From the right labyrinth arise tonus to those muscles producing movements towards the right as well as toward the left, but the stronger impulses are those which produce movements toward the left. The tonus for the two sides exactly balance each other and a state of equilibrium results. An increase or a decrease in the tonus from one side would upset this equilibrium and result in vertigo. This is observed in its most pronounced form for the sudden destruction of one labyrinth. Now the tonus from the opposite normal ear throws the patient toward the diseased side and a violent rotating nystagmus develops, with the pull or slow component towards the diseased side. How compensation is restored and equilibrium reestablished cannot be discussed here.

Phenomena of Fatigue.—Under this heading I shall discuss three sets of phenomena resulting from the rotation experiment. The first is the facts brought out by the early work of

Barany, namely, that the maximum nystagmus following rotation develops after approximately ten rotations and that a shorter nystagmus develops either after a shorter or longer rotation. This phenomenon finds a ready explanation as a phenomenon of fatigue, if our hypothesis is correct, regarding the duration of the endolymph currents on starting and stopping rotation. The explanation is as follows: In the rotation experiment we recognize a much more violent stimulation of the end organs than we meet with in our daily experience. It is to be expected therefore that phenomena of fatigue might appear. When we rotate a patient toward the right there develops a nystagmus toward the right caused by the inertia of the endolymph, and this nystagmus continues so long as does the peripheral stimulation—that is, for approximately ten rotations. If now we stop rotation at the moment when the peripheral stimulation occasioned by the rotation ceases, we will get the maximum after response, for the reason that impulses emanating from the opposing groups of hair cells, on stopping rotation which produce nystagmus to the left, are not impeded by tonus from the hair cells stimulated during rotation, since these are in a state of fatigue. Suppose that the turning is stopped after but five rotations—that is, before the maximum stimulation has been developed from these hair cells actively stimulated during the turning, then the after nystagmus must be weakened because of the opposing tonus from those cells stimulated during rotation but which are not as much exhausted as they must be after ten rotations. Suppose again that instead of stopping at the end of ten rotations—that is, at the moment when the peripheral stimulation ceases—we continue the turning for 50 or 100 rotations, during all this time there will be no stimulation of the hair cells going on, for the reason that the endolymph is moving as fast as the canals. During all this extreme rotation the hair cells stimulated during the first ten rotations are recovering from their fatigue. The result will be the same as after five rotations, a shortened after nystagmus.

The second phenomenon of fatigue is the following: It has been observed that the moment the nystagmus which developed during rotation ceased, there may develop for a few moments a nystagmus towards the opposite side, although the

turning is going on at the same pace. The explanation as a phenomenon of fatigue is quite apparent. At the moment the nystagmus created by active rotation ceases, that moment the endolymph current also ceases, and the tonus from the hair cells which would be stimulated on stopping rotation out-balance for a few moments the tonus for those cells actively stimulated during rotation; the result will be the development of a nystagmus towards the opposite side exactly as though the rate of rotation had diminished or the turning had been stopped entirely.

There is still one other rather interesting phenomenon, the explanation of which comes in the same category as the phenomena of fatigue from overstimulation of the peripheral mechanism. This is a phenomenon described by Barany in his earlier observations on vestibular stimulation. He found that in individuals, where the response was particularly acute, that if, instead of stopping the turning at the end of ten rotations the turning was kept up for a considerable period—that is, for 50 or 100 rotations—that on stopping rotation the following phenomenon developed: First an after nystagmus of shorter duration than that which would result had the turning been stopped at the end of ten rotations. At the moment when this after after-nystagmus ceased there developed for a few moments an after nystagmus directed toward the side toward which the patient had been turned. Applying the same principles just gone over, the explanation as a phenomenon of fatigue is clear. In the first place, all we need to assume is that the active stimulation of the hair cells causing a nystagmus toward the same side as the rotation ceases after approximately ten rotations. Now the rotation is continued long enough to permit of a complete recovery from the fatigue occasioned by the violent active stimulation during rotation; then on stopping rotation the response elicited by the stimulation of the opposite group of hair cells producing nystagmus toward the opposite side will be somewhat weakened because of the vigorous tonus from those cells which were stimulated during rotation but which had been given ample time for recovery before the rotation was stopped. In exactly the same way those cells stimulated by the inertia of the endolymph on stopping rotation are being vigorously overstimulated, so

that when the after nystagmus ceases—that is, when the endolymph movement entirely disappears—these cells are in a state of fatigue so that the vigorous tonus from the opposing group of cells, lacking for a few moments the balance from the opposite groups of cells, now produce the phenomenon of after after-nystagmus as described by Barany. This explanation may on first thought seem a little involved, but it is in reality extremely simple, and it appears to be involved only because of the roundabout method which one must use in describing the several reactions. A graphic representation of the two horizontal canals shows the thing very quickly and with great simplicity.

A NEW MAGNIFYING GLASS FOR OTOSCOPY.

BY ALEXANDER ZEBROWSKI, M. D.,

NEW YORK.

The need of obtaining clear and at the same time magnified otoscopic pictures was felt at the very beginning of otoscopy. In the year 1873 Trautmann¹ described the manner by which a common magnifying glass (a biconvex lens) can be used for otoscopy. For this purpose Trautmann screwed the lens into the ear speculum, changing, when necessary, both the glass and the speculum. Still earlier, in the year 1823, the magnifying glass had been used by many otologists, among others Deleau and Buchanan. Bonnafont (1834) used a compound magnifying glass of three lenses.

Afterwards Broeck, Cleland, Weber-Liel and others used a lens.² Politzer³ and Brühl⁴ advocate the use of a lens for otoscopy, and both emphasize the great importance of obtaining clear, distinct otoscopic pictures. Well known is a lens of Brünings by means of which we can very easily get a magnified view of the drum and of all possible pathologic changes in the latter. Brünings' lens is of great advantage, especially for beginners and for the otoscopy of normal drum membranes.

To my knowledge the latest magnifying glass described and recommended for our profession is an apparatus of Prof. Dr. Demetriades of Athens (Greece),⁵ consisting of a metallic lug on which are placed two metallic grooves. One groove is for the lens, the other for the ear speculum. The lens has a triangular opening which enables the use of a probe and little surgical instruments while looking through the magnifying glass.

In the year 1918, at the session of the Austrian Otological Society,⁶ I demonstrated an ear magnifying glass which for three years I used continually in otoscopy, and which has rendered me valuable services. My magnifying glass differs from the others, because it is not placed in the aperture of the reflector or on the ear speculum, but is attached by means of a

half ring to the first phalanx of the left thumb (Fig. 1). By means of a double joint the magnifying glass can be turned in every direction. The lens can be easily removed from the metallic groove and replaced by the other. We can use the lenses with all possible focal distances. I give the focal distances of the lenses which can be used for otoscopy:

Diopters	Focal distance
13 D.....	77 mm.
12 D.....	83 mm.
11 D.....	90 mm.
10 D.....	100 mm.

Personally the most suitable lens for my eye is 11 D. focal distance 90 mm. The lens is not round, but on one side a segment has been cut away (Fig. 2). It does not interfere with obtaining magnified otoscopic pictures, but enables the aurist (besides the use of the probe) to perform under the control of a lens several small operations (irrigation of the attic, intratympanal otoscopy, removal of small polyps, granulations, etc.). In the after treatment of the radical mastoid operation the magnifying glass can be very useful, because it makes it impossible to overlook exuberant granulations, diseased areas of the bone and accumulations of even the smallest quantity of pus. The discovery and treatment sometimes of small and hidden perforations in the Shrapnell membrane becomes much easier with the use of a lens than with the naked eye.

The magnifying glass described has been made by Reiner⁷ (Vienna) and is to be obtained there.

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Fig. 1.

Magnifying glass in position. Use of the probe.

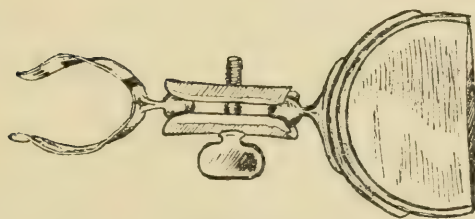


Fig. 2.

Magnifying glass. Double joint.

XXXVI.

OTITIS MEDIA COMPLICATING OPERATIONS ON THE GASSERIAN GANGLION.

By H. R. LYONS, M. D.,

ROCHESTER, MINN.

Otitis media complicating operations on the gasserian ganglion was first observed in the Mayo Clinic about three years ago, in a patient who complained of a sense of fullness and deafness, following section of the posterior root of the gasserian ganglion for trifacial neuralgia. Since then such observations have been made several times. This type of otitis media occurs from a few days to several weeks after operation on the gasserian ganglion. The chief complaint is a sense of fullness and deafness on the side on which the operation is performed. Otalgia is never severe and is not a common complaint. Examination of the ears discloses a fullness in the inferior quadrants of the tympanic membrane, with more or less obliteration of the common landmarks. There is usually a fluid level, and a bubbling sound is heard with inflation of the eustachian tube. Two patients said that they had "water in the ear." The tympanic membranes were pale in each instance. In this condition there also appear within the middle ear bleblike formations without evidences that the remainder of the cavity contains fluid. The tympanic membrane is gray or pink and does not contain generalized fluid. It is flaccid or atrophic in appearance, is not intensely red, and never has the sense of resistance with the otoscope. Deafness, as tested by tuning forks, was of the conduction type and not severe. The posterior superior wall of the canal did not droop.

In classifying the pathologic conditions in the middle ear in cases of otitis media, the question arises whether the fluid is exudate or transudate. Because of the close association and connections of the tympanic plexus in the mucous membrane of the middle ear with the branches from the gasserian ganglion, as described here, it is definitely established that the fluid is the result of an altered condition of the mucous mem-

brane consequent to the operation. It is accepted that the operation results in a trophic disturbance in the mucous membrane of the middle ear, causing the collection of fluid. I believe that this type of otitis media is due to the secretion of the fluid, but the point cannot be definitely established. Animal experimentation as a means of investigation might have been feasible, except that the results of section of the posterior root of the gasserian ganglion in animals not suffering from trifacial neuralgia would not be parallel to those following a similar operation on patients with a definite disturbance of the sensory root, or of the gasserian ganglion, as in the cases here reported. It was therefore concluded that such investigation would not be of value.

In one case observed (Case 1), secretory otitis media followed alcohol injection of the posterior root of the ganglia. Spontaneous rupture and secondary infection, resulting in suppurative otitis media, occurred. It is also of interest that the posterior root was injected, since the neuralgia disappeared at once and cutaneous anesthesia followed.

In another case of secretory otitis media, not reported because the condition occurred several weeks after the patient went home and our information was not definite, suppuration occurred, but cleared up promptly. This man had a corneal ulcer, as did one other patient.

Abrasions of the cornea are not uncommon following operations on the gasserian ganglion; two of this series of patients had corneal ulcers. I am unable to determine whether such ulcers are due to trauma or purely to trophic local changes. Both causes may exist and possibly a combination of the two, namely, lowered local resistance due to the trophic changes, with subsequent trauma from dust and other foreign materials. Although goggles of the automobile type are worn by such patients postoperatively, trauma cannot be ruled out definitely.

In many respects the picture of otitis media, due to trophic disturbances following gasserian ganglion operations, simulates herpes zoster otiticus, differing chiefly, however, in that pain is absent in the former and severe in the latter; also the bleb formations on the pinna are absent in this form of otitis media.

NERVE CONNECTIONS.

Sensory and trophic nerve supply to the mucous membrane of the middle ear is abundant and is directly connected with the sensory root of the gasserian ganglion. The tympanic plexus in the middle ear also has direct connection with the posterior root of the fifth cranial nerve, the sphenopalatine ganglion, and the cervical sympathetic nerves.

The sensory nerves to the external auditory canal are derived from the auriculotemporal branch of the trigeminus and from the auricular branch of the pneumogastric. The latter, also known as Arnold's nerve, perforates the wall of the meatus and supplies its lining membrane.

The posterior wall is separated from the mastoid process by the tympanomastoid fissure. The auricular branch of the pneumogastric nerve passes through this fissure to the posterior wall of the canal. The coughing, sneezing or vomiting that sometimes follows irritation of the canal, as from cleaning the ear or examining it with instruments, is said to be due to a reflex effect on the pneumogastric nerve through this branch. The auriculotemporal branch of the trigeminus nerve enters into its supply and may explain the earache in cases of cancer of the tongue or disease of the lower teeth.

The nerve supply of the tympanic membrane is derived chiefly from the auriculotemporal branch, supplemented by twigs from the tympanic plexus and by the auricular branch of the vagus. They accompany, for the most part, the blood vessels and, besides supplying the latter, form a subcutaneous and a submucous plexus. The tensor tympani and tensor palati muscles receive their nerve supply from the same source, namely, the trigeminus, through the otic ganglion.

The nerves supplying the mucous membrane of the tympanum are branches from the tympanic plexus formed by the tympanic branch of the glossopharyngeal nerve, in conjunction with sympathetic filaments from the network accompanying the carotid artery. The tensor tympani muscle receives its supply from the trigeminus, the stapedius from the facial. Although the chorda tympani nerve has an intimate topographic relation to the space which it traverses close to the outer wall, it gives no filaments to the structures within the

tympanum. The nerves of the eustachian tube are supplied from the tympanic plexus and from the pharyngeal branches of the sphenopalatine ganglion.

The tympanic nerve, or Jacobson's nerve, arises from the petrous ganglion and traverses a tiny canal in the osseous bridge between the jugular fossa and the carotid canal. Entering the tympanic cavity and receiving fibers from the carotid plexus of the sympathetic by way of the small deep petrosal, the tympanic nerve passes upward and forward in a groove on the promontory and breaks up in this situation to form the tympanic plexus. After distributing filaments to the mucous membrane lining, the tympanic cavity and the associated air spaces (mastoid cells and eustachian tube), its fibers re-assemble and join with a filament from the geniculate ganglion to continue as the small superficial petrosal nerve to the otic ganglion.

The branches of the tympanic nerve are: The small superficial petrosal nerve, the branch to the fenestra ovalis, the branch to the fenestra rotunda, the branch to the eustachian tube, the branch to the mastoid cells, and the branch to the great superficial petrosal nerve.

The auricular branch of the vagus is given off from the ganglion of the root. It receives a filament of communication from the petrous ganglion of the ninth nerve and follows the outer margin of the jugular foramen to an opening between the stylomastoid and jugular foramina. Entering this foramen, it traverses a canal in the temporal bone which crosses the inner side of the facial canal and terminates between the mastoid process and the external auditory meatus. Leaving the canal, the nerve supplies the skin of the posterior part of the auricle and of the posterior inferior portion of the external auditory meatus. While traversing the temporal bone, the auricular nerve communicates with the facial and, after reaching its arc of distribution, with the posterior auricular nerve.

The communicating branch of the facial nerve to the tympanic plexus traverses a tiny canal in the temporal bone to reach the tympanic cavity where it joins the main continuation of the tympanic plexus of the glossopharyngeal to form the small superficial petrosal and proceeds to the otic ganglion, which it enters, as a sensory root. The fibers from the tym-

panic plexus, probably secretory in function, are distributed from the otic ganglion to the parotid gland.

REPORT OF CASES.

Case 1 (A398947).—Mr. S. A. H., aged forty-eight years, came to the clinic complaining of severe pain throughout the left side of his face, which was recognized as trifacial neuralgia. July 28, 1922, the patient was relieved by a preliminary alcohol injection of the sensory root of the fifth cranial nerve. He returned in about two weeks because he had a recurrence of the severe pain. Examination of his ears was negative. August 10, a gasserian ganglion operation was performed on the left side, the posterior root being cut. Three days after operation the patient complained that his left ear felt full; this feeling persisted, with slight deafness.

Examination showed the right ear to be normal; the left tympanic membrane was pale, the lower quadrants bulging and the upper normal. Motion of the tympanic membrane with an otoscope confirmed the patient's suspicion that there was water in his ear, because the fluid could be seen to move. Changing the position of the head changed the relation of the fluid level to the ear landmarks. Eustachian tube inflation caused a bubbling sound. Functional examination of the hearing revealed a slight conduction type of deafness. There was no otalgia at any time. Paracentesis was not necessary, and the patient was dismissed from observation three days later. In a letter, August 18, he says that symptoms of fullness and deafness are decreasing. The fluid level is lower.

Case 2 (A359235).—Mr. W. B., aged 48 years, had typical trifacial neuralgia. His ears were normal. Three alcohol injections were given on the right side May 28, December 15, and December 19, 1921, respectively. The last injection afforded relief, but the patient had a sense of fullness and deafness in the right ear. The ear ruptured spontaneously, and a thin watery fluid escaped. No further treatment was necessary. December 29, 1921, a gasserian ganglion operation was performed, the right sensory root being sectioned. Three days later, before the primary dressing was changed, the patient complained of slight pain in the right ear, soon followed by a spontaneous discharge of clear fluid.

Examination revealed a pale tympanic membrane slightly full in the lower quadrants with a posterior inferior perforation, and a thin fluid pulsating through the opening. This clear fluid became definitely purulent on the following day by secondary infection. Slow but steady improvement then occurred. January 14, 1922, when the patient was dismissed from observation, the tympanic membrane was retracted, the perforation healed, and subjective and objective symptoms had entirely disappeared.

Case 3 (A365379).—F. N. B., aged sixty-five years, had trifacial neuralgia. Preoperative examinations revealed normal ears. July 30, 1921, the left sensory root of the gasserian ganglion was sectioned. About nine days later the patient complained of fullness and slight deafness in the left ear.

The typical pale membrane with slight fullness in the lower quadrants and the slight deafness typical of the condition were present. There was no otalgia; paracentesis was not considered necessary. The patient was seen daily and the ear rapidly cleared. This patient also had a corneal ulcer in the left eye which was typical in its resistance to local treatment. Further trauma was eliminated by the use of a glass eye shield.

Case 4 (A358945).—Mr. T. E. G., aged sixty-eight years, had a right gasserian ganglion operation for trifacial neuralgia May 19, 1921. The sensory root was cut. The findings in the ears were negative. The patient was dismissed from observation without complications. However, a letter from his home physician revealed that he had subsequently developed right otitis media which spontaneously ruptured and became secondarily infected. He also developed a right corneal ulcer. Both resisted local treatment. A later letter, December 19, 1921, stated that both the ear and the eye were about cured.

Four other cases belong to this series, but since a personal examination was not made they are not incorporated in this report. The patient in Case 1 was typical in every detail and most interesting. It should be noted, however, that all the patients had complete relief from the pain after operation; they also had cutaneous anesthesia over the surface supplied by the posterior root of the ganglion. It should further be noted that two of the four patients had trophic disturbances

in the form of secretory otitis media and corneal ulcer; this again brings up the question of the factor of trauma in these cases of postoperative corneal ulcers.

SUMMARY.

1. Secretory otitis media following operations on the gasserian ganglion is an entity. It is probably due to trophic disturbances in the mucous membrane of the middle ear.

2. The nerve connections between the gasserian ganglion and the mucous membrane of the middle ear are abundant.

3. The ear and eye complications are always on the side on which the operation is performed.

4. Trauma, as an etiologic factor, is eliminated so far as the ear is concerned, and this gives further basis to the argument that the corneal complications are entirely trophic in origin.

5. The process may be similar to that occurring in a herpes zoster otiticus.

XXXVII.

NASAL REFLEX.*

BY ROBERT F. RIDPATH, M. D.,

PHILADELPHIA.

Nasal reflex or neurosis as a cause or in conjunction with other symptoms has been and still is in many instances a puzzling mystery; that located on or within the Schneiderian membrane there is a reflex to practically every part of the body has been demonstrated by many authors and writers, but to my knowledge has never been compiled into one paper or subjected to a dissertation under one heading.

It is therefore with this end in view I have made a compilation from the literature of the past several years, and have added thereto my personal experiences, in the hope that this subject may be made available for future reference.

INNERVATION OF THE NOSE.

The innervation of the nose is from the olfactory bulb sending filaments to the upper one-third of the nasal mucosa through the cribriform plate; from the sphenopalatine ganglion sending fibers to the superior, inferior and middle turbinates and to the septum, and from the external twig of the nasal nerve to the anterior part of the outer wall of the nasal chamber.

An important point to be borne in mind is that the mucous membrane is endowed with general sensation through the fifth nerve, by which vasomotor control is exerted in the vascular mechanism underling it.

The nerve connections from the nose to the genital sphere are especially numerous from the turbinated bone, as also those from the nose to the stomach from the middle turbinated bone, and from the nose to the heart from septum place.

F. Blumenthal¹ in critical studies of the nasal reflex neurosis says: "Irritation of the centripetal nerve fibers of the nasal

*Accepted candidate's thesis, American Laryngological, Rhinological and Otological Society, January, 1923.

mucous membrane causes functional disturbances in the centrifugal nerve region, and the sympathetic fibers are brought into play, which in the majority of cases may be considered as the nerve tract. These sympathetic fibers are numerous distributed in the nasal mucous membrane, and are secretory but here are vasomotor."

One can therefore imagine that the different reflex arcs which extend from a single organ are differently localized and that they do not run into each other but meet the nasal mucous membrane in certain places. Thus can the existence of the genital, gastric and cardiac places be explained.

B. F. Andrews² in 1915 states that "Intranasal stimuli may produce coughing and sneezing, which is doubtless brought about by impulse being switched onto the vagus, which receives sensation from the pharynx, larynx, bronchi and lungs, and motion to the muscles of the voice production, and some respiratory muscles in the larynx. It is fair to assume, then, that intranasal irritation is responsible for at least a percentage of cases of asthma associated with hay fever."

The nasal reflexes which are generally considered physiologic are sneezing, lacrimation, various secretions, turgescence, cough and hyperosmia.

Jurasz³ divides the disturbances of the normal reflex processes into three groups. In the first group are those in which the irritation affects the nose and also affects an additional reflex action beyond the nose, as asthma, spasm of the glottis, nightmare, nervous cardiac troubles, as cardiac pain, tachycardia, angina pectoris, epiphora, disturbances in the ears, migraine, headaches, epilepsy, exophthalmic goiter, St. Vitus dance, nocturnal enuresis, melancholia and hysteria. The second group includes the nasal reflex neurosis in which the sensory irritation exists in the nose and shows itself through a reflex in the nose such as hay fever, rose cold, railway and ipecacuanha cold. Finally among the third group are the reflex neurosis in which the sensitive irritation takes its origin in another organ and may transfer the reflex to the nose. There are again certain kinds of nervous colds, bleeding from the nose, sneezing, hyperemia and swelling of the nasal mucous membrane.

J. P. Lopez,⁴ in his paper "New Reflex Symptoms of the Nasal Origin," gives reflex mydriasis as caused by nasal obstruction, noting four cases in 20,000 patients, and states: "It has been found to coincide with the intense nasal obstructions, and the proof that these attacks of mydriasis are derived from nasal obstructions lies in the fact that mere touches of cocaine in the fossæ are sufficient temporarily to suppress the anisocoria, which disappears completely after an operation which corrects the nasal obstruction. The symptoms are rarely noticed by the patient, for in general it has no harmful sequela."

The writer concludes that in spasmodic mydriasis the reflex is transmitted through the intervention of the great sympathetic which governs pupillary dilatation; the reflex, starting from the nasal fossæ, whose nerves, forming ends in the sympathetic, would be excited by the inflammation of certain zones of the mucosa, causing the reflex in the ophthalmic ganglion to determine the mydriasis by means of the dilator nerves of the pupil which proceed from the ganglion.

The exact examination of Killian⁵ showed that in the region of the extension of the ethmoid nerve in the anterior section of the nose not all the mucous membrane regions were the place of the reflex actions in question. The point in which an increased sensitiveness in contrast to fine tactile irritation with great regularity could be proved was the tubercula septi and the section of the mucous membrane situated over the anterior ends of the inferior turbinated bones. In the normal, the author was successful in separating these places, through a great irritation, from epiphora and nose irritation.

ASTHMATIC REFLEX.

Asthma as a nasal reflex has been considered and recognized by numerous observers. Sluder⁶ says: "There is a great difference of opinion as to the way the impulse is transferred from the cervical sympathetic to the cardioaccelerator and pulmonary vasomotor fibers, but it seems that the relay station for the asthma reflex must be in the cervical sympathetic ganglions. If the asthma reflex may be controlled from the nose ganglion or the sphenoid sinus, then there should be places from which the reflex may be started experimentally or accidentally. Reflex asthma gives a decidedly different aspect

from the cardiac asthma or the asthma dyspepticum. In the typical asthma attack there are besides dyspnea in the expiratory phase, which produces flatulence of the lungs, the falling of the diaphragm, catarrh of the lungs and discharge containing Charcot-Leyden crystals and Curschmann's spirals, which make sure the diagnosis.

Kuttner⁷ characterizes asthma as a genuine reflex neurosis and concludes that "Here (in asthma) we have the type of a pathologic reflex in which the irritation of the reflex center is greater than norm, and the region of excitation is extended above the norm, and as the anatomic substratum in this pathologic reflex process is unknown at present, so we have to consider the reflex asthma as a genuine reflex neurosis."

The author wishes to report a case of asthma of true nasal reflex.

Mrs. J. P. K., age fifty-four, white, married, has had three children, all living and well. She had asthmatic attacks for fourteen years, occurring with equal violence night or day. She was referred to me by a diagnostician after years of treatment for intestinal toxemia, cardiac arrhythmia and unnumbered other diagnoses and treatment had utterly failed to have the least curative effect. Wassermann negative; blood pressure, 138-90. Well formed woman of middle age. Examination of nasal fossa showed a high deviation of the nasal septum pressing on middle turbinate right side, vomer ridge in left fossa forming synechia with posterior end of inferior turbinate. There were no polypi or other pathologic condition in the nose. During the preliminary examination (which was made without cocain) the examining probe, when brought into contact with the synechia, caused a violent attack of asthma, lasting thirty-five minutes, and was only relieved when the nasal mucosa was thoroughly cocainized. A submucous resection of the nasal septum was performed, separating the synechia and relieving the pressure of the middle turbinate, with absolute alleviation of her asthmatic attacks for the past eight months.

CARDIAC REFLEX.

Rugani Luigi,⁸ in *Arch. Ital. di Otol.*, etc., 1920, states: "It is a well known fact that a not inconsiderable number of

individuals affected with cardiac rapidity are relieved from military service after fruitless efforts at cure and after passing the period of reexamination. It is therefore to the recognition of the fact that cardiac rapidity is consequent upon a nervous reflex of nasal origin and that even today little or no value is attached to the etiology of such disease of the cardiac apparatus."

E. T. von Bucke⁹ in his numerous experiments coincides with others and concludes that "The insufflation of irritating gases or fumes into the nose, with an expiratory stopping of breathing, produces an interference with the activity of the heart, which in the first place may be considered as a reflex irritation of the vagus through the sensory branch of the fifth cranial nerve to the nasal mucous membrane."

Knoll found that the retarding of the action of the heart with the irritation of the nasal mucous membrane in rabbits could not exclusively be traced back to an irritation of the cardioinhibitory fibers of the vagus.

Von Brucke reports seven cases (rabbits) in which the pulse frequency decreased during the chloroform experiment. In this respect H. Roger,¹⁰ in his admirable paper, suggests that the brisk projection of irritative vapors (ammonia, and especially chloroform and ether) into the nasal cavities stops respiration during expiration and raises the blood pressure, which is followed by systodiastolic oscillations. The cardiac manifestations are the same, even when the animal (rabbit) is submitted to artificial respiration. The brisk projection of irritant vapors through a tracheal tube produces respiratory spasms, with marked dilation of the thorax, which contracts very slowly. The cardiac manifestations are the same as in the irritation of the nasal mucous membrane. The cardiac manifestations are due to simultaneous action of two antagonistic powers, one lowering and the other raising the pressure. The first depends on the pneumogastric. After double vagotomy the neocardiac reflex is simplified, the depressive phase is omitted. The excitation of the nasal or bronchial mucous membrane brings at once a rise of pressure. In some cases irritation of the nasal mucosa causes a tetanic contraction of the heart.

Inhaling chloroform or ether does not produce the same effects as the brisk projection does; it lessens the respiratory movements and lowers the blood pressure, which seems to be due to toxic absorption.

DIFFICULTY IN DEGLUTITION.

F. Pontoppidan¹¹ reports two cases, the first in which deglutition was interfered with, and the second, that of persistent headache. I quote the first, which to me has more bearing as to a reflex cause than the second. "Female, forty-three years old, referred to the author for trouble in swallowing. She was unable to swallow potatoes, meat or rye bread. She lost weight. Pharynx showed slight yellow secretion from the nose. No headache but a feeling of pressure at the bridge of the nose. Diagnosis: Ethmoiditis duplex. Partial resection of the ethmoid cells. Removal of polypi. Patient recovered and can now swallow all food easily. Doubtless this was a case of reflex spasm of the constrictors pharyngei caused by the nasal condition."

NAUSEA.

Greenfield Sluder in 1921 observed and following numerous others have verified his observations that many cases of persistent and otherwise unaccounted attacks of nausea may be the direct result of a nasal reflex. Sluder's¹² observations were brought about when injecting the sphenopalatine (Meckel's) ganglion with 95 per cent alcohol. Nausea was produced and vomiting continued for six days intermittently. The same phenomenon also follows the postethmoidphenoid operation. After adding carbolic acid to the alcohol injection, nausea is much less frequent, but still sometimes follows.

Frequently pain from any cause produces nausea and vomiting. On many occasions a severe nasal ganglion neuralgia has been accompanied by nausea, which ceased with the cessation of pain by anesthetizing the ganglion. The author relates one case of severe nasal neuralgia which was accompanied by vomiting, even when pain was absent. The application of one drop of 10 per cent cocain to the nasal ganglion district stopped the vomiting. In another case of hyperplastic non-suppurative sphenoid headache, marked nausea without head-

ache is sometimes manifest. Applications of one drop of 10 per cent solution of cocain to the floor of the sphenoid cell stops it in ten minutes.

The author believes that these two anatomic subdivisions of cases should be thought of as one type, namely, that of sympathetic nervous system irritations, because of the intra-sphenoid cocainization of the vidian nerve in the vidian canal.

SEXUAL REFLEX.

Many authors, Fleisz, Killian, etc., have demonstrated beyond doubt the points of the nasal sexual reflex; confirmation has been the result in the hands of unnumbered rhinologists. These places are first the anterior position of the two lower turbinated bones, and second, the two sides of the nasal septum opposite. These show two elevations richly supplied with nerves and blood vessels, the so-called tubercula septi.

Fleisz, according to his reports through cocain experiments, could absolutely state to which group of the dysmenorrhea the single case belongs, for in the nasal form of the same he was always "dead certain" to remove the pain through cocain application in the genital places of the nose, and vice versa. After the effect of the cocain had ceased he could again kill the pain with applications of trichloracetic acid.

Skillern¹³ in his work, "The Accessory Sinuses of the Nose," states that "Inflammation of the sinuses often exercises a marked influence over the sexual function. In ordinary cases there is a marked deterioration, while in the severe cases it may be totally abolished."

One can readily see that the toxemia taking place during a sinusitis will lessen sexual inclination and function, and if the discharge continually pours and covers the points already proven it finally destroys the sexual nasal reflex. Of several cases I will report the following: Miss M. B., age 18, white, well developed and perfectly healthy except prior and during menstruation. Mother stated that two days before the flow she always complained of a fullness in the nose with a corresponding difficulty in breathing, also a terrific headache and cramps. These were of such severity that the patient was compelled to seek her bed, where she remained four days. During this time there was no alleviation of head or abdominal pains

except through medication. She had consulted two gynecologists and had two curretments without any relief. The last gynecologist, Dr. Brinkmann, sent her to me for nasal examination. Examination at this time revealed nothing abnormal. As this was midway between the menstruation, advised her coming a few days before the next periodicity.

On second examination, made three days before the expected flow, I found both inferior turbinates swollen to such an extent as to occlude the inferior nasal passage and press on the septum. On cocainizing, the mucous membrane of both turbinates contracted and looked perfectly normal. The patient was suffering from no other nasal or other condition that I could elicit.

Prescribing a 1 per cent cocain solution and advising the patient's mother to allow a few drops in each nostril several times a day and to report to me in two days, I was surprised, knowing the history of the case, to have the patient present herself for further examination and treatment on the day specified, stating that there had been no pain or other symptoms, although the menstruation had now been of twenty-four hours' duration.

Future treatment consisted in destroying the nasal sexual reflex areas by cautery, and the patient has had no return of her former discomfort for three years.

HAY FEVER

As the literature pertaining to hay fever neurosis is so voluminous and easily obtained, I shall not go into this subject. Sufficient to say papers and articles by Hallopeter, L. B. Baldwin, A. H. W. Caulfield, of Toronto, Nicholas and Lillian Kopeloff, A. B. Conklin, Frank Parker, of Stockbridge, George W. McKenzie and many others all clearly demonstrate the reflex neurosis in this disease.

HYPEROSMIA.

Increased sensitiveness to detect odors is found in the temperamental, hysterical, epileptic, hypochondria and may result from any invitation of the olfactory bulbs.

PAROSMIA.

Parosmia with subjective perception of imaginary odors, according to Sir St. Clair Thompson,¹⁴ is always a central nerve affection.

Patient reported by the author at the Philadelphia Laryngological Society in 1918: T. B., man 42, was referred to the Medicochirurgical Dispensary from the Neurological Department for complete examination of the upper air passages. Patient states that "he is always pursued by a horrible odor," this seeming to have its origin in his own body. While traveling on trolley cars, he invariably rides on the front or back platform so as not to disturb or be disagreeable to other passengers, and even in this position he is so conscious of the odor he frequently leaves the car to take another, perhaps several times before arriving at his destination. He also frequently changes boarding houses for fear the landlady will find out his peculiarity. Asked "if anyone ever spoke to him of it," he replied: "No, that seems to be the peculiar part; nobody knows I suffer from such a disagreeable disease except myself, and no one seems to be able to smell the odor but myself, but I am afraid they will." He would not visit his relatives or friends for the same reason.

Examination of nasal cavities revealed nothing abnormal, and after several visits to the dispensary vanished from our observation. Although apparently normal in other respects, we are forced to agree with F. St. John Bullen¹⁵ that parosmia is a perversion of the olfactory sense and frequently occurs in the hysterical and insane.

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XXXVIII.

TUMORS OF THE JAW FROM THE STANDPOINT OF THE RHINOLOGIST.

By J. D. WHITHAM, M. D.,

NEW YORK.

Although new growths of the jaws are relatively uncommon, nevertheless their early recognition and early treatment are matters of importance. Malignant neoplasms of the jaws grow into the accessory sinuses and toward the base of the skull. Scudder states that "an intimate knowledge of these sinuses is necessary to the operating surgeon." The rhinologist has this knowledge and should possess the ability to diagnose and treat these conditions.

In the few minutes allotted, I shall attempt to briefly discuss the more frequent neoplasms of the jaw, giving the salient points in diagnosis and treatment.

An epulis is probably the most common tumor of the jaw. It is a connective tissue growth seated on the border of the alveolar process of the jaw about the teeth. It is seldom larger than an olive and is usually covered by intact mucous membrane. It nearly always occurs in young adults. Two varieties are described; the fibrous and the more common giant-celled type. The latter is softer, more vascular and more irregular in shape than the former. Metastases do not occur but the disease is locally malignant. The condition is treated by extracting a tooth on each side of the tumor, notching the bone in front and behind and removing the alveolar process with growth attached by a chisel. When this is done the condition never recurs.

Sarcoma of the jaw is extremely rare in childhood and when seen is usually of the giant-cell type. Most frequently it is found between the ages of 15 and 60. Trauma is believed to be an important etiologic factor. It is more frequent in the lower jaw and is apparently about equally as common in the jaws as carcinoma. A rare but very malignant form origi-

nates from a blood vessel. This is called a perithelioma. The two types of sarcoma most frequently seen are the central or medullary type originating from the bone marrow, and the peripheral or periosteal type, which, as its name implies, grows from the periosteum. The medullary type is surrounded at first by a definite shell of bone and periosteum. Often it is difficult to distinguish such a growth from a benign cystic tumor. The tumor growing rapidly destroys the bone, breaks through its capsule and invades the soft parts of the face or neck. The peripheral type, growing from the periosteum, does not cause extensive destruction of bone, and is not surrounded by a definite bony shell. It is softer than the bone to which it feels firmly attached and has definite outlines. In both jaws the body and alveolar processes are attacked with equal frequency.

The symptoms of sarcoma of the upper jaw are a swelling of the jaw with evidences of necrosis of bone; tenderness along the alveolar border; a tender spot on the cheek; loosening of teeth; ulceration extending to the hard palate; obstruction of one or both nasal cavities from a growth often resembling a nasal polyp; a continuous foul purulent nasal discharge with intermittent nasal hemorrhages; a tingling and numbness of the upper lip and cheek; a flattening and later a bulging of the hard palate; diplopia; edema of the eyelids and face; empyema of the maxillary sinus and evidences of a progressive necrosis of the jaw. The rhinologist is often called on early in these cases. With the aid of the X-ray and his experience he is especially qualified to make an early diagnosis. In reaching a diagnosis the following conditions must be excluded; alveolar periostitis, gumma, dental cyst, carcinoma and odontoma.

The treatment of sarcoma of the jaw usually entails an extensive primary operation, although in less malignant types a limited operation may be undertaken. The advantage of using the actual cautery in removing these growths is well established. The use of radium and X-ray in treatment will be briefly considered when carcinoma of the jaw is discussed. The results of all forms of treatment are so bad and the mortality so high that very early diagnosis seems to offer almost the only hope. By studying and thinking more about these

conditions and by co-operation with the oral surgeon, rhinologists can do much.

Benign tumors of the jaw are usually of the following types: fibroma, myxoma, osteoma, chondroma and mixed growths. The sinuses are frequently involved. Such tumors are slow growing but often produce most repulsive deformities of the face. The treatment is, of course, entirely surgical.

Another type of jaw tumor which is relatively common and which should be thoroughly understood is the odontoma. An odontoma arises from a portion of a tooth follicle or tooth germ as the follicle is sometimes called. To understand how this occurs, a rapid review of the development of a tooth is necessary. A tooth, like a hair, develops in early fetal life from an invagination of the epithelial layer into the mesoderm. In this way a flask shaped epithelial cord is formed which is called the enamel organ. The mesoderm of the rudimentary jaw grows upward and is called the papilla. This becomes completely surrounded by the enamel organ which is, in turn, surrounded by the sac of the tooth. The sac, the enamel organ and the dentin papilla together comprise the tooth follicle.

From the enamel organ of epithelial origin grows the enamel of the tooth. When certain of these epithelial cells of the enamel organ in the root of the tooth cease to form enamel, they may remain as epithelial cell rests. It is from these cell rests that the adamantine epithelioma or odontoma is believed to grow. This is a slow growing immovable tumor. It is more frequent in the lower jaw and is occasionally cystic. The patient is usually a young adult. This growth is locally malignant. One of its most interesting features is its microscopic resemblance to a carcinoma. The treatment is entirely surgical. If any doubt exists as to its malignancy, a resection should be performed. No case of recurrence after complete removal has ever been reported.

Cysts of the jaw are classed among the odontomata as they also grow from the tooth follicle. These are of importance to the rhinologist, especially when occurring in the upper jaw. They are of two kinds—the periosteal, or root cyst, and the follicular or dentigerous cyst.

The root cyst is the more frequent. It arises probably from the epithelial sheath of the enamel organ, beginning as a granuloma at the root of a tooth. A periodontitis ensues with degeneration and softening of the granulation tissue and cyst formation. The fluid is a clear yellow and contains cholesterin and cast-off epithelial cells. In the upper jaw, growing in the line of least resistance, it may encroach upon the antrum or upon the mouth or nasal cavity. In the lower jaw it is seen on the lingual aspect, under the mucous membrane. The teeth are often displaced to one side. Fluctuation and the parchment-like crackling may be obtained. In the upper jaw, the floor of the nose and the floor of the antrum may be displaced upward. Exploratory puncture and the X-ray aid in diagnosis and are usually necessary when an infected root cyst has perforated into the antrum. The treatment is best carried on by the Partsch operation. This consists of a complete removal of the oral wall of the cyst and opens the cavity fully into the mouth. It always cures the condition.

The follicular or dentigerous cyst, though not so frequently encountered as the root cyst, is nevertheless a common form of benign odontoma. In the upper jaw it occurs as an olive-sized swelling on the outer side of alveolar process. It does not seem to encroach upon the lingual side. Its wall feels thin, and on palpation the parchment-like crackling may be felt. It appears usually during or shortly after the second dentition. It often develops from an unerupted canine or molar tooth, and the absence of this tooth may be noted. A dentigerous cyst arises from the follicle of an unerupted tooth, and at operation one or more tooth-like objects will be found in its cavity. It is usually monolocular and may grow to great size. The lower jaw in the molar region is the most frequent seat. Treatment is entirely surgical and consists of a complete removal of one wall of the cyst, thorough curettage of the interior of the cavity, and packing with iodoform gauze. This can usually be done through the mouth, and in this way little, if any, deformity will result.

The last type of jaw tumor to be considered is carcinoma. This will be spoken of briefly, for it is a subject quite familiar to us all. It is four times more frequent in men than in

women. In the jaws it is almost always primary. The central carcinoma of the upper jaw is nearly always primary in the maxillary sinus, and is usually of the cylindric cell type. More commonly, however, carcinoma begins in the oral or nasal mucous membranes. When the disease begins in the nose or sinuses, polypi or polypoid growths are usually present. A bloody discharge from one nostril, with nasal obstruction, should always excite suspicion. Neuralgic pain in the cheek is often an early symptom. Bulging of the antral wall, apparent in the cheek, orbit or nasal cavity is a later symptom. Enlargement of the cervical glands is a late manifestation, especially when the upper jaw is the seat of the disease. When the antrum is attacked a purulent sinusitis is nearly always associated. The X-ray is invaluable in the diagnosis of this condition.

After operation the average time for recurrence, according to Martens, is nine to ten months. It has been reported, however, as late as after eleven years. The results after operation are even worse than is the case with sarcoma. The use of radium and X-ray with early resection by the actual cautery seems to offer more hope. The treatment of an operable case of carcinoma of the jaw should be, in brief, as follows: About two weeks of preliminary intense treatment by radium should be given, using bare tubes and packs. During this period the teeth should either be extracted or carefully scaled and cleansed. The nose, throat and mouth should be cleaned every few hours with a mild spray and wash. The patient should be made accustomed to the stomach tube, that he may be less disturbed by its use after the operation. Very light ether anesthesia by the intratracheal method, with careful packing of the pharynx, will be found essential. A preliminary ligation of the external carotid artery on one or both sides is usually advisable. Everything should be in readiness to compress the common carotid artery if this step should later become necessary. The Rose position is believed by many operators to be the best. A suction apparatus is extremely helpful. The resection of large growths is best done by the cautery. Following operation, the patient should be fed by the stomach tube for about seven days, and external X-radiation should be given.

My object in reporting the following case is to show the possibility of removing one-half of the mandible without causing a very conspicuous deformity and to emphasize the usefulness of a preliminary ligation of the external carotid artery. In hemiresection of the mandible there is a great loss of blood when the ligation is omitted, despite the dexterity of the surgeon. The ligation serves to minimize the loss of blood, injury to the parts in manipulation and time of operation.

Patient, K. F., age 45, female. This woman has been an inmate of the King's Park State Hospital since 1902. She is feeble-minded.

Family, social and previous medical history have no important bearing on the case. In December, 1921, it was noticed that the patient was developing a swelling on the left side of the mandible. It was thought at first that this was a dento-alveolar abscess, and the molar teeth were removed. The tumor, however, continued to increase in size, and by February, 1922, had extensively involved the alveolar mucous membrane which was ulcerated. On February 19th, a section of the growth was removed. This was shown to three pathologists. One said the growth was a carcinoma, and two called it a spindle celled sarcoma. On May 14, 1922, a hemiresection of the mandible was performed under light chloroform anesthesia. The left external carotid artery was first ligated distal to the superior thyroid artery. This wound was carefully sutured and sealed with cotton and collodion to prevent contamination during the subsequent operative procedures. A vertical incision was then made on the lower jaw in the midline, commencing just below the carmine of the lip and extending downward to below the inferior border of the jaw, severing the soft parts, to the bone. The lower end of this incision was then extended backward below the jaw to the angle. The right coronary artery was clamped and ligated. The tumor was carefully separated from the surrounding soft parts. The levator menti, platysma and buccinator muscles were severed by the knife from their attachments to the bone, and while the anesthetist retracted the cheek the bone was cut from the oral mucous membrane. It was not possible to save the alveolar mucous membrane, as it was involved in the growth. The

massiter muscle and stylomandibular ligament were next divided with scissors and the mandible was cut through with a saw in the mid line. The cut end of the bone was then grasped with large bone forceps and forcibly depressed. This manipulation exposed the tendon of the temporal muscle at its attachment. Here it was cut with scissors, taking pains to hug the bone to avoid injury to Steno's duct. The external and internal pterygoids were next cut and the bone with tumor attached was readily removed by twisting and cutting the capsular ligament with blunt scissors. Up to this point the operation had been almost bloodless. Now a slight venous ooze from the pterygoid plexus was noticed, which was readily controlled by the gauze pack. All glands which could be found were removed. The oral mucous membrane was closed by a chain stitch of chromic catgut, the massiter and pterygoid muscles sutured together and a drain of one foot of iodoform gauze was inserted. The skin was closed, leaving an opening for drainage. The time of operation was one hour and forty-five minutes. The patient made a rapid afebrile recovery. She has had no return of the growth, has gained in weight and does not appear to suffer any inconvenience in eating as a result of the operation.

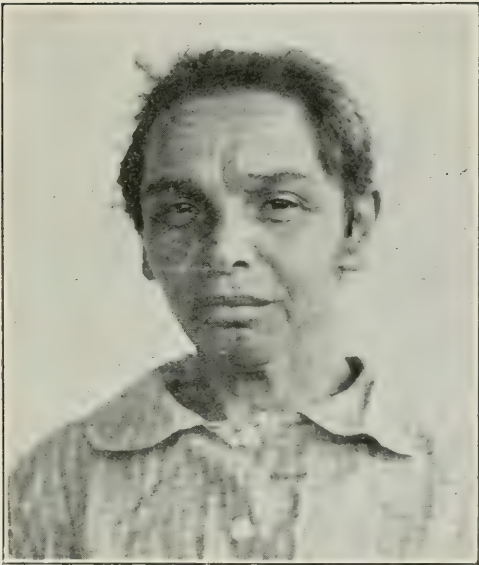
The growth and slide were shown to Dr. James Ewing, who called it an adamantine odontoma. The alveolar arrangement of the epithelial cells and the anastomosing stellate cells are characteristic.

The report of the pathologists before operation left the surgeon no choice. A complete operation was indicated.

34 EAST 61st STREET.



K. F. Adamantine Odontoma. Before Operation.



K. F. Adamantine Odontoma. After Operation.

XXXIX.

PHLEBOTHROMBOSIS OF THE INTRACRANIAL SINUSES.

BY C. COULTER CHARLTON, M. D.,

ATLANTIC CITY, N. J.

As early as 1816 Abercrombie¹ drew attention to thrombosis of the lateral sinus, giving the symptoms and pathologic observations. Many articles have been written since then, but especially in the past twenty years most work has been done along this line. Knapp,² Van Dusch,³ Lapersonne,⁴ and Coupland⁵ were the first to give the profession valuable knowledge concerning thrombosis of the cavernous sinus, the signs of which are largely ophthalmic.

In 1845 Bouchut⁶ brought forward the results produced by marasmus in the coagulation of the blood in the veins, especially of the extremities, and stated that it may also be the cause of thrombosis of the intracranial sinuses.

The great width, the shape, the rigidity of the sinus walls and the angle, often against the current, at which the veins empty into the sinuses, all tend to favor coagulation in these localities.

Dental caries which caused a suppurative phlebitis, as well as gangrenous tonsils,⁷ have been reported as causes of sinus thrombosis; but the inflammatory or infected form, which mainly interests us, has as its most frequent cause suppuration of the middle ear and the destructive processes arising from it; yet any pathologic condition about the head or face as well as trauma may cause thrombosis. Anthrax, erysipelas, orbital cellulitis, retropharyngeal abscess as well as the above ear conditions have been reported as causing thrombosis of the intracranial sinuses.

In infective thrombosis there is always a phlebitic condition which, on account of the inflamed vessel wall, causes the blood to adhere and clot. This clot generally disintegrates rapidly as germ laden pus formed by the vessel wall mingles with it and forms greenish brown fluid which is filled with living

organisms and is likely to be carried into the circulation with widespread metastases.

The clot in sinus thrombosis begins by a fibrin deposit at point of invasion of the infection and may gradually enlarge to obstruct the vessel and extend from the lower end of jugular vein to all its tributaries, facial, thyroid, condylar, mastoid, emissary, superior and inferior petrosals, and through the lateral to the sinuses of opposite side. These thrombi may be mural or complete. When complete the extremities may be sterile through the action of the antiseptic circulating blood and yet the center, if infected, remains so. When the infected portion breaks through into the blood stream, septic emboli are carried to the lungs, joints and superficial tissues in the order named. Well presented, as the subject has been by Whiting and others, there is little need to go into the symptoms of sinus thrombosis, but it may be well to give a few which serve to differentiate the sinus involved.

The superior longitudinal sinus is most frequently affected with marasmus thrombosis, but being superficial and connecting with the parietal, the diploe and the emissary vein of Santorini makes thrombosis liable to occur from trauma and inflammation of scalp.

Edema of the scalp, overdilatation of the veins over the parietal and the occipital regions, occasionally over the frontal when the anterior portion is involved, slight degree of squint are frequently observed and epistaxis on account of venous connection with nose, especially in infancy and childhood.

The Cavernous Sinus.—In the beginning of thrombosis of this sinus, the symptoms may be unilateral, but in over 50 per cent of cases it becomes bilateral. When the other side begins to show involvement, the one first observed may begin to show improvement of symptoms. The two groups of symptoms that are typical in cavernous sinus thrombosis are those due to obstruction of the venous circulation and to paralysis due to pressure on the nerves surrounding or passing through the cavernous venous plexus. The first group comprises exophthalmos, edema of the eyelids and the root of the nose, chemosis of the conjunctiva and retinal changes from engorgement of its veins; the second group strabismus, ptosis and

pupillary disturbances, which show involvement of the third, fourth and sixth nerves.

The Sigmoid Sinus.—Of all the sinuses the one that lies nearest to the cellular structure of the ear is the sigmoid portion of the lateral sinus. On the left it carries most of the blood from the interior of the brain, while on the right side it drains the surface blood from the veins. It varies in distance from the suprameatal spine, from close proximity to about one inch, and is the one most frequently involved. Körner states on an average the right sigmoid sinus is about 2.55 mm. nearer the postmeatal wall than the left.

Sigmoid invasion generally arises from a middle ear involvement. If in a chronic otitis media there is a sudden cessation of discharge with persistent pain extending into the head, high temperature with steeplelike fluctuation, vomiting and rigors, it may be regarded as pointing to thrombosis of the sigmoid sinus. Other symptoms that may be looked for are venous obstruction of the sinus, which causes overdilation of the external jugular and other superficial veins, and those caused by mastoid inflammation, tenderness and edema back in the posterior cervical triangle from the emissary vein as it emerges at the occipitotemporal suture. There may be enlarged cervical glands, especially along the internal jugular and soft tissue, cervical abscesses arising from venous thrombosis, systemic affection, pulmonary involvement, an infected enteritis and metastatic invasion of the joints or other tissues, even in the remote parts of the body.

The first record in otologic literature of operation on the lateral sinus is that performed by Samuel Ashhurst of Philadelphia about 1875. Since then Zaufal, Horsley, Ballance, Lane, Grunert and Whiting have been the most influential of the pioneers in the field, while McKernon, Randall, Day and other American otologists have added a large contribution of cases to the list. In a careful study of the lists that have been reported and published, together with my limited experience, I think a great many of them were only phlebitis cases without thrombosis. A great number of these cases would no doubt have responded with less operative interference and many that died might have recovered if it had not been for the shock to the patients during the operation. This point

has been ably brought out by Randall.⁹ Quoting from his paper, he says: "The point which I have long insisted upon, that essential differences exist between the cases due to acute otitis as contrasted with the chronic, seems increasingly substantiated; but I believe that we must try better to recognize the nonthrombotic cases which may do better without operation."

Of course, we all know that a man of Randall's ability may do such, but for the average otologist, I am afraid, his patient would be submitted to a far greater risk than an exploration of the sinus, whether it be due to phlebitis or a phlebothrombosis. Many times the jugular has been ligated and sinus opened, even where no clot was found, and a fatal result avoided, as the bacteremia caused by phlebitis had been cut off from the general circulation.

Many nonfebrile cases have been reported by Alexander,¹⁰ Wild,¹¹ Uchermann,¹² Uffenorde¹³ and others where the sinus was obliterated. Ewing Day¹⁴ reported six cases of spontaneous cure accidentally discovered during operation. So it behooves us to go carefully and use every means to study our cases most thoroughly so as to give our patients the advantage of nature's own protective powers without lowering the resistance unnecessarily with operative procedures.

The consensus of opinion among otologists is in favor of ligation of internal jugular vein for lateral sinus thrombosis. I might state that, according to published records of many of the cases where the ligation was performed, no clot was found, but many recoveries took place because they were cases of phlebitis with bacteremia and by ligating infection was cut off from the blood stream.

Therefore, each case must be given a careful study, for, while we must realize that it is very important to operate early, it should not be at the sacrifice of our patient.

In the severe type of phlebothrombotic cases operated on about 80 per cent of the cases are successful, while in the milder type only about 70 per cent are successful.

The uncovering of the sinus presents little danger, but the statement made by some that the sinus wall would not be injured if the chisel or curette is used carefully is not altogether true, according to reports, as well as my own observation.

Grunert and Zeroni,¹⁵ Panse¹⁶ and Körner¹⁷ have reported grave sequelæ from exposure of sinus wall.

When the sinus has been exposed it should be packed with iodoform gauze plugs so that the pressure will be between the bony structure and soft tissue wall, completely compressing the vessel. After this the sinus should be opened between the plugs and the connected contents evacuated. After evacuation of the sinus thoroughly one plug is removed, and if spontaneous bleeding takes place it should be replugged, then the same thing should be done to the other end. If free bleeding does not take place search should be extended for the cause and curettement continued until bleeding is established. These plugs should not be removed before eight or ten days; if there is no bleeding replugging is unnecessary. A large portion of sinus wall should be removed.

In summarizing I wish to emphasize the following points:

1. All cases of suspected phlebothrombosis of the cranial sinuses, whether acute, chronic, traumatic or marasmic, should be carefully studied.

2. When we do operate, it should be done rapidly, thoroughly and with as little manipulation as possible.

3. Shock should be combatted by applying heat locally, hypodermoclysis and intravenous administration of saline and adrenalin when indicated.

4. We must strive better to recognize the nonthrombotic cases which might do better without operation.

5. Let us all try to develop our skill to such a degree that we will not submit our patients to any more than is absolutely necessary for a successful recovery.

CASE REPORTS.

Case 1. W. R. is a boy 12 years of age; the family history and that of the patient are negative, so far as the present case is concerned.

History of case: Two weeks previously he had been bathing in a pool one evening, and during the night complained of an earache. Next day he was all right and went to school. The family physician was called later to see the boy with a history of having chills and fever for three days. His report shows

that he found boy lying on his back with legs flexed; his eyes were closed and oversensitive to light, the pupils equal but slow in reaction to light (there was no nystagmus) and he did not complain of headache. The breathing was rapid; the temperature was 103 F.; there was slight stiffness of the muscles of the neck and the reflexes were exaggerated. The patient was very irritable when moved or otherwise disturbed, but quiet when left alone.

The following day I was called in consultation and found the above mentioned symptoms present, just as stated by the family physician, except that temperature was now higher, being 104 F., the pulse 124 and the respirations 24. On examination I found the nose and throat apparently normal, as were also the tympanic membranes; there was no tenderness or swelling over the mastoid regions or over the remaining portions of skull.

A blood examination was made, showing: Hemoglobin, 80 per cent; red blood count, 4,000,000; white blood count, 20,000; differential count, normal; urinalysis, negative.

A spinal puncture was made and the fluid, which flowed under a slightly increased pressure, was clear. Microscopic examination showed about six leucocytes to the field, and cultures taken, after seventy-two hours, were negative.

The patient was seen by me on three consecutive days before I could convince myself that the case was anything more than one of meningitis. On the third day I was able to detect a small localized area of tenderness over the right mastoid posteriorly, but other physicians who were present were inclined to doubt this.

As the patient was getting worse and the case seemed hopeless unless something was done, I advised operation. The patient was sent to the hospital and the mastoid opened. The outer plate was hard, but the cells and the inner plate soft, and after curetting, the sinus was exposed and pus found surrounding it. The sinus wall looked healthy, was compressible, pulsating and soft, no indication for opening. The patient was returned to bed with a fair prognosis.

By the fourth day after operation the patient's temperature was normal and remained so for about seven days, when it suddenly rose to 104 F. The day before this rise the patient

had complained of tenderness and swelling in the left elbow, which on the following day had to be put in a splint on account of the extreme pain on the slightest movement of the joint. A culture was made of the blood, but it proved negative. X-ray plates also failed to show anything. Cultures from the wound proved the presence of a mixed infection, showing staphylococcus pyogenes albus and a few short chains of streptococci.

A few days after the involvement of the elbow the patient's left ankle became red, swollen and painful. The swelling was so great that aspiration was decided upon and a ropy fluid was obtained, but no growths appeared on cultures made from this material. X-ray plates of this joint were negative. At this time the temperature jumped up to 105 F. and I decided to open the exposed sinus, which bled very freely, but a small thrombus was obtained. In this case the jugular was not ligated. Within three days the temperature was down to 100 F. and he gradually improved. It was necessary to keep the ankle and elbow in splints. After the temperature had remained normal one week the ankle was put in a plaster cast and the patient was able to get around on crutches. It was six weeks before the elbow cleared up; the ankle took longer, two months elapsing before the patient could give up the use of his crutches, and three months before the ankle was normal.

Case 2: E. R., a boy of 9, was first seen at my office on January 8, 1920. His mother brought him in for an examination, as he had been sick frequently with upset stomach and as she thought not as strong and healthy as he should be. He gave a history of having his tonsils and adenoids removed four months previously. About one month before he came to me he had a sore throat, slight fever and complained of both ears. At that time, his mother stated, he had white spots on each side of his throat. He was sick only a few days and everything apparently cleared up. Two weeks later the doctor who treated him for the above attack was called again. At this time, his mother said, he had the same kind of spots on his throat, but also had a slight rash on his body. The health physician of our city was called in consultation, as they thought it might be scarlet fever, and it would be necessary to quarantine him. After consultation it was decided he did

not have scarlatina and he was treated for a sore throat. After a few days he seemed to be entirely all right.

Examination.—The boy was apparently well formed and well nourished. His throat showed pieces of tonsils that had not been entirely removed, containing small crypts filled with cheesy debris, but about a normal color. The ears at the time were apparently normal except for a slight dull thick look to the membrane tympani, about what would be expected from a child that gave a history of frequent attacks of tonsillitis and sore throat. Not being able to find much wrong with him in my line of special work, I referred him to a pediatrician of our city, who reported to me as follows:

Pediatrician's Report.—E. R. History of convulsive attacks after any illness accompanying fever. Fairly well. Sore throat and fever five days at time when supposed to have scarlet fever. Slight efflorescence not followed by desquamation, abscess over body frequently lately, headache, no running of ear. Appetite fair, bowels good, restless at night. All organs normal, no enlarged bronchial glands. Reflexes normal. The attacks are like epilepsy, but always with fever. Gave instructions as to diet. Ten days later records show patient recovering from tonsillitis. Earache; gets pale and face twitches, probably convulsive attacks, as he always has fever. Physician ordered sodium bromid, five grains, t. i. d.

The pediatrician asked me to see the boy again and I found him suffering from an acute follicular tonsillitis. At this time his ears did not give him any trouble, but I looked at them each time I saw him, but did not observe anything wrong. In a few days his throat had cleared up and I did not see him again until about the 23rd of January. At this time he had complained during the night of a fullness and slight pain in left ear. I examined him carefully; his temperature was 101 F., pulse 80, respiration 20, throat and nose slightly congested, ears apparently normal, hearing good. For the next three days I saw him daily and he remained about the same except an increase in temperature to 102 F. At this time the boy looked ill, and on account of the history of some pain in left ear I did a paracentesis. Why I did this I do not know, as I was not able to observe enough to warrant it; perhaps remembering the custom at a children's hospital where I formerly

did some work of puncturing the drum if a child had a fever and if it could not be accounted for. In this case only the one was opened in which he had previously complained of pain. The paracentesis was performed at 6 P. M. January 25th, and only about one drop of blood was noticed. Later in the evening I made arrangements with one of our general physicians to see the case the next day. Early the next morning I called to see patient and found him with every symptom of acute mastoiditis and a profuse discharge. He was sent to hospital; laboratory examination made of blood showed: Hemoglobin, 88 per cent; red blood count, 5,000,000; white blood count, 12,800, polynuclears, 78 per cent; lymphocytes, 26 per cent; large mononuclears, 5 per cent. Temperature at this time was 103 F., pulse 104, respiration 28. One hour later his temperature jumped to 104 F. and an operation was decided as soon as patient could be prepared. A simple mastoid operation was performed.

Operation Findings.—Thick outer wall of mastoid, cells partly broken down; pus; necrosis; soft bone and pus; sinus exposed about one and one-half inches; was pulsating and compressible; wound left open and dressed. Patient sent to his room. Next morning temperature was 100.3-5 F. and remained there for three days. Suddenly on fourth day after operation temperature rose to 104 F. Patient developed a rash on right arm and chest, was restless, meningeal cry, semi-conscious, Kernig's sign present, stiffness of neck and tenderness of joints with pain on slightest motion or pressure.

The visiting ophthalmologist at the hospital examined his eyes and reported partially choked disc and that in a few hours he would no doubt be blind.

Lumbar puncture was made at this time. Fluid clear, but came out under great pressure. Culture made and laboratory reported a few chains of streptococci.

After spinal puncture eyes examined next morning and doctor was surprised at the way the eyes had cleared up.

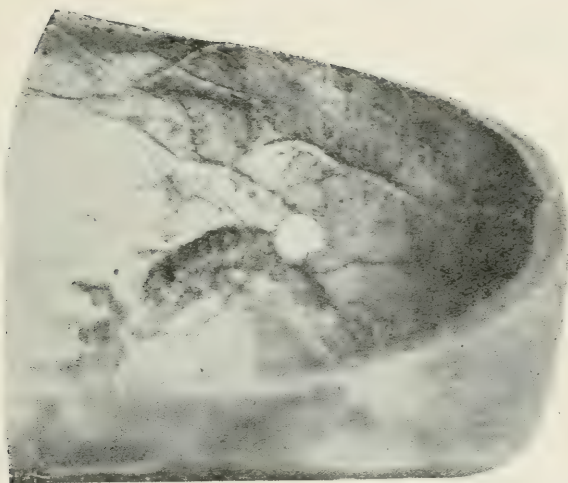
Patient was sent to the operating room, as he had another decided chill and we realized something had to be done rapidly to save the boy. Granulations were cleaned out, sinus exposed and opened, no blood from proximal end. The jugular vein was ligated and a portion resected, sinus then curetted

and a large thrombus was found down in the bulb. Distal end being packed off with iodoform gauze, a cigarette drain was inserted into the bulb and dressing applied. After three days the temperature was down to 100° F., and gradually improved till patient was transferred to his home, seventeen days after operation.

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124 SOUTH ILLINOIS AVENUE.



A skull trephined for mastoid caries and lateral sinus thrombosis
by the late Dr. Samuel Ashhurst of Philadelphia,
some forty years ago. (8)

XL.

DENTAL RELATIONS OF THE EYE, EAR, NOSE AND THROAT.

BY W. H. HASKIN, M. D.,

NEW YORK.

These relations are far more numerous than are generally recognized, and should be considered under several heads. There is scarcely time to give more than a brief outline of some of the most important.

The most prominently before us at the present time are those conditions arising from focal infections, but as a matter of fact they should be the last to be considered if we learn to study diseases from the standpoint of cause and effect.

The writer feels that this subject is of far greater importance than that simply dealing with focal infections, and that for it to be fully appreciated one must go back even to the early embryonic stage of development. Many of the serious problems start at this time, and can be recognized in the fetal skulls at birth, as will be shown on the screen from slides loaned by Dr. B. W. Weinberger, of New York, who has studied and written extensively of his findings.

The following outline is suggested to anyone who cares to take up a real study of the subject, and it is a most fascinating one, growing more interesting the deeper one goes into it.

1. Maxillary development.—(a) Embryology of bones; (b) embryology of teeth and alveoli; (c) postnatal growth; (d) faulty developments and causes.

2. Faulty maxillary development.—(a) High and narrow arches; (b) short anteriorposterior arches; (c) narrow pyramiform orifices; (d) dental irregularities, over and underbite; (e) delayed dentitions and impactions; (f) effects of premature extractions; (g) probable effect on the orbital cavity causing heterophorias; (h) atresia of the posterior choanæ.

3. Causes of faulty development.—(a) Systemic diseases of parents; (b) improper balance of foods, vitamin; (c) insufficient endocrine secretion; (d) insufficient amniotic fluid in

utero; (e) artificial bottle feeding; (f) insufficient mastication, i. e., lack of proper stimulation of tissues to induce growth, both of teeth and bone; (g) premature extractions.

4. Nerve reflexes due to faulty developments.—(a) Headaches not localized; (b) pains referred to ears, eyes, etc.; (c) neurasthenias and insanity; (d) dysphagia and dysphonia; (e) tinnitus aurium; (f) eyestrain; (g) general irritability.

5. Focal infections.—(a) Apical abscesses, acute and chronic, toxemias, bacterial infections; (b) pyorrhea.

6. Causes of pyorrhea.—Uncleanliness, poor mouth hygiene; accumulation of food particles, improper brushing, poor contact points. Malocclusions, faulty occlusal surfaces; supra and infraocclusions, uneven surfaces of tooth at cervical margins. Traumas, rough edges of fillings; marginal injuries, rough edges of crown bands; rough edges of cavities; ceruminal calculus; improper use of toothpicks. Systemic disturbances: Syphilis, diabetes, tuberculosis, anemias, scurvy and rickets, intestinal toxemias, metallic poisonings, improper diet.

7. Maxillary tumors.—Sarcomas, malignant sarcomas, non-malignant fibromas, myxomas and fibromyxomas, dentigerous cysts, cysts from apical abscesses, osteomas, odontomas, supernumerary teeth, polypi.

Time will only allow me merely to touch upon some of the conditions that occur during the different periods as classified. Failure of union between the frontonasal process and the maxillæ, either on one or both sides, results about the tenth week of fetal life. The dental laminæ appear on the alveolar processes of the maxillæ as early as the seventh week and calcification during the sixteenth week. The crowns of all the deciduous teeth, the four permanent incisors and the four first molars have already reached their final size at the time of birth and distinct evidence of faulty structure is found in them even before they erupt, these faults leading to early decays and premature loss. It has been well established that malocclusions are often found at birth, due, it is claimed, to deficient amniotic fluid while in utero and in some cases to the traumatism at delivery. These malocclusions are so marked in some cases as to interfere with proper nursing and the bottle has been resorted to for that reason and not from any lack of milk supply on the mother's part. It is in-

teresting to note that enlarged adenoids are present in these cases.

In the second classification the nose and throat are directly affected very seriously and all because of the teeth and indirectly the ears are very likely to suffer secondarily. Failure of the teeth to spread the upper jaws causes the high palatal arch generally associated with angular deviations of the septum, especially when there is a delayed eruption of the upper permanent incisors, as shown by Dr. Mosher, and these narrowed jaws will generally show a great narrowing of the pyriform orifices. It is at this point nasal obstruction will be most commonly found, and this also constitutes the reason for so many failures to relieve obstructed breathing in even the most satisfactory submucous operations. Every nose should be carefully examined for this point of obstruction before operation, and if it is present it can be safely said that there is a malocclusion. If it is present you may be sure that there is a malocclusion with a narrow palatal arch, the upper teeth occluding within the lower, so that every effort of mastication only tends to increase the narrowing of the upper arch instead of tending to spread it if they were in proper occlusion. On the other hand, the arch may be plenty wide enough but there has not been enough downward and forward growth due to slow development from inadequate stimulation of mastication (this being probably the greatest factor in producing such appalling early decays in the teeth of our children). In these cases you will find a marked vertical bow in the septum, which is probably explained by recognizing that the vomer belongs to the cranial bones and grows to correspond in size with them, only to find that the maxillæ have not grown sufficiently and that there consequently is not room for the vomer and it bends as a result. Also this failure of forward growth interferes with the caudal rotation (Dr. Schaeffer) of the posterior end of the alveolar arch and causes impactions of the molars, and undoubtedly reacts upon the nerves which descend on the posterior wall of the antra. It thus affects the innervation of the eyes, which with the shortening of the orbital cavity in all probability can account for many of the heterophorias which are commonly found at this period of the child's development, as observed by Dr. Daly of Boston several years ago.

In the writer's experience, delayed dentition has been found the cause of many serious ills, both painful and otherwise, and it has become a routine practice for him to examine all patients for these easily determined conditions, and to be guided by what is found. There are two periods where delayed growth can readily be determined: First, whenever it is found that the distance between the inner cusps of the child's second molars does not measure 33 mm. there has been insufficient growth, and if this distance is less than 29 mm. at three years, Dr. Bogue claimed that there never would be sufficient growth without artificial aid. This can be supplied by systematic daily exercise of the muscles of mastication, as suggested by Dr. Rogers of Boston, but it takes a great deal of patience on the part of whoever is in charge of the child. Second, if the deciduous incisors fail to separate before the fifth year there will be delayed dentition of the permanent teeth, not only of the incisors, but also of all the teeth, for the upper ten permanent teeth occupy the space left when the deciduous teeth are thrown off, and the separation of the deciduous teeth must take place if the arch is to become large enough for the permanent teeth to erupt in a natural manner. As mentioned before, Dr. Mosher claims that delayed eruption of the upper incisors is a frequent cause of septal deflections, the premaxillary wings being displaced to one side or the other. There are many other important things to be considered in thinking of these cases, but time will not allow me to describe them at this reading, but I want to add this important fact, and that is that in all cases of delayed dentition the roots of the teeth are bound to be distorted in the same direction, and this adds to the difficulties encountered later in their treatment. It is also a fact that these cases are always associated with some form of malnutrition and with poorly developed chests and lungs. A great deal has been written on the influence of the endocrine secretions as a contributing cause of delayed dentition, and it is claimed that the lack of these secretions in the milk of bottle fed babies causes faulty growth of the teeth from lack of proper calcium deposit, thus accounting for the rapid decays in early life.

Another group of investigators have clearly shown how a lack of the proper balance of the vitamins will produce decal-

cification of both bone and tooth structure in animals and that by giving the proper balance the calcium content will quickly return to normal.

One could write a long paper on the last two phases alone, and much that is of great interest and value has already been written on the subject by Howe, McCollum, Griener and others.

Personally, the writer believes that the greatest of all causes in producing malocclusions with the resulting asymmetrical developments of the face, causing nasal obstructions and orbital troubles, is the lack of full muscular efforts in feeding. This begins in infancy and continues on practically throughout life, especially in this country. One can readily appreciate the muscular action that goes on in a sucking child's mouth by inserting a finger in his mouth. Dr. M. Hellman has pointed out the great loss that occurs in the bottle fed babies, when he states that the average time consumed by an infant in emptying an eight ounce bottle provided with the best nipple and having the smallest aperture is about eight minutes, which with eight feedings a day would give only sixty-four minutes of actual work in the twenty-four hours, whereas the time consumed at a breast feeding averages about twenty minutes, or one hundred and sixty minutes, a difference of nearly one hundred minutes.

When one stops to consider that the growth of bone depends largely upon the stimulation supplied by muscular action, he is forced to appreciate what such a great difference must mean in the facial development.

Dr. Hellman further states that of eighty-four cases of malocclusion personally studied, 81 per cent occurred in bottle fed children.

Dr. Herdlicka, in a paper from which the writer quoted in a paper written in 1915, states that after an observation of 960 children of Apache and Pima Indians, lasting over two years, there was no abnormal narrowing of the maxillary arch nor protrusion of the incisors, and no irregularities, except an occasional crowding of the incisors, and that the arches were broader in the second temporary molar region than in the case of white children with the same type of skull and that the bones were generally heavier. That he never found a case of adenoids or of tonsillitis in an Indian child nor one who

was a mouth breather, this in spite of the fact that they lived in the worst possible hygienic atmosphere of dirt and poor ventilation. He also made this most significant statement: The Indians have had and still have the habit of keeping up the nursing of the child until the second or third year, and even later. And this important fact was noted, that the mother gave her child various things to chew upon very early in life while she still continued nursing late.

Here we have the double value of muscular action and a natural sterile and easily digested food, supplemented at an early period by food that required mastication, and thus produced stimulation of the dental germs, thus aiding in producing strong, healthy teeth with firm alveolar processes to hold them in place.

Another most significant fact is that the immigrants of all countries entering this country, with the exception of the English, Irish and Norwegians, have remarkably well developed jaws and teeth, attributed to the foods that they have eaten and their habit of nursing their young.

The exceptions use the refined flour breads, large quantities of potatoes and well cooked cereals, none of which requires much mastication.

Again, the children of these same immigrants very quickly begin to show the same dental disturbances that are found in our children, probably due to the change in food and consequent loss of muscle stimulation.

What all this has to do with the dental relations of the ear, nose and throat will be readily realized when its scope is understood.

Every pronounced case of malocclusion is a mouth breather and is thus more susceptible to colds and sore throats, with the possibility of infections of the ears. Mouth breathers lose the great moulding power of the tongue in spreading the arches both outwards and forwards, dragging the arch downwards, and the facial muscles are constantly at work pulling the bony structures in abnormal positions. The teeth being poorly developed, rapidly decay and are prematurely lost, adding another factor in delaying eruption of the second teeth, and throwing the whole balance of articulation out of gear when the sixth year molars are lost, for they are the real

anchors of development of the jaws during the crucial period when the deciduous teeth are being thrown off and the permanent cuspids and bicuspid are erupting. When it is known that among the high school children of New York, averaging about 14 years, less than one-half of 1 per cent were found to have four permanent first molars and that in many extensive decays were found in one or more of the teeth, it will be understood how important it is for all medical men to make it their duty to watch the teeth of all their small patients and insist upon their care.

As stated at the beginning of this paper, focal infections hold the place of greatest interest in the minds of most of us. I now want you all to think the above rambling remarks over carefully, for if we could only make the parents take that interest in their children and learn how much depends upon the proper foods and mastication of these foods, we would go a long way towards preventing malocclusions and early decays. In this way there would be a great difference in the development of the abscesses which are so universally found, and in the dreaded cases of pyorrhea, which are ever on the increase, due to the poor nutrition, the irregularities, the fillings, crowns and other forms of dental restorations. There is hardly a journal published in these days that does not contain an article on focal infections or refer to them in some way, and I shall not attempt to tell of my own years of experience, but will close with a heartfelt plea for you all to study it all out for yourselves to realize how important it is to get down to actual causes if you are fully to appreciate what the effects are to be and how you can best combat them. The damage has already been done beyond repair in most of our mouths, but we ought to be able to stop a great deal of similar troubles in our children if the effort is made intelligently, and if the generations following us could only carry on the work it would not be too much to hope for to have a stronger and better developed race of people besides the saving of the suffering that comes from diseased teeth.

XLI.

THE CORRECTION OF EXTERNAL DEFORMITIES OF THE NOSE.

BY SIDNEY ISRAEL, M. D.,

HOUSTON, TEXAS.

Reviewing the necessity for, and the accomplishments of, plastic surgery during the recent war, directed at various parts of the human anatomy, and recreating, so to speak, many unfortunate persons, one can well appreciate the reason why rhinoplastic surgery has attained its present interest in the field of corrective or cosmetic measures, in an effort to transform one of the most noticeable features of the face, when unsightly, into something more useful and agreeable to look on.

It is of sufficient import to mention, in passing, the mental unrest in the minds of certain of these patients, resulting from the extreme self-consciousness of an unsightly feature of the face.

Historically, this character of surgery originated many years before, but its application and refinement of technic, together with numerous original ideas, have developed during our time, as a result of the investigation of such enthusiastic workers as Joseph of Berlin, Major Gillies of England and our own Roe, Carter and Cohen, and others.

Etiologically, external deformities of the nose should be classified into two main groups: (1) Congenital, and (2) acquired; the second group, including those due (a) to trauma, or (b) to disease (syphilis, lupus, etc.).

Anatomically, or from the parts involved, these deformities may consist of: (1) Bone, (2) cartilage, or (3) bone and cartilage.

In directing our attention to the correction of these deformities, several essential principles are to be kept in mind, if our results are to be all that we would have them:

1. There must be no external or visible scar, if it can possibly be avoided (the so-called intranasal approach).

2. We must have a clear understanding of the deformity and the necessary measures for correction, suitable to the individual face. This can be gained only after careful study and planning prior to operation.

3. Our technic must be as mechanically and aseptically above criticism as possible.

4. Whatever surgical step is taken must be corrective and accurate.

With these essentials, we turn our attention to the various types of external deformities of the nose, usually encountered in civil practice: Hump (dorsal); lateral bend; drooping or elongated tip, with or without dorsal hump, the so-called hook nose; pug or upturned tip; wide nostril with flat tip (African type); and saddle deformity, or a combination of one or more of these.

We find, in many cases of external deformity, an associated deviation of the nasal septum, requiring correction. I agree with other observers that only a conservative form of septal resection should be performed, to avoid failure or mishap.

Local anesthesia is the one of choice wherever it can be employed, and its use offers many advantages that are only too obvious. This form of anesthesia has been made use of even in the correction of saddle deformities of the nose, wherein a piece of the costal cartilage was taken for transplantation. When the technic of local anesthesia is properly carried out, there should be no pain. The patient is placed in the sitting position during operation, or semirecumbent at an angle of 45 degrees.

The technic of operation is as follows: After the preliminary cleansing of the face, the skin over the dorsum and sides of the nose is painted with a 5 per cent solution of tincture of iodine. The nostrils are cleansed with warm physiologic sodium chlorid solution, and the anterior nares swabbed with a 5 per cent solution of tincture of iodine. The line of incision is made within the vestibule in the outer nasal wall, through the cartilage, until the skin covering the nose is reached. Through this incision, repeated on the opposite side if necessary, the skin and periosteum over the entire nose is elevated from the nasofrontal articulation to the tip, and as far over the nose laterally as required.

With this subcutaneous elevation of the skin accomplished, through the incision, whatever surgical procedure necessary to properly correct the existing deformity, whether it be the removal of a hump, straightening a lateral bend, elevating the tip for the correction of hump nose, or the reception of a cartilage transplant, can be readily carried out, with no injury to the overlying skin and the avoidance of external scar.

In order to avoid an external scar, the incision should be made within the nose, the so-called intranasal approach. I cannot see why any of these patients, possessing no defect of vision, should be required to wear spectacles with plano lenses, merely to hide an external scar over the bridge of the nose that could have been avoided by the intranasal approach.

In the use of transplants for the correction of saddle deformities of the nose, cartilage has in our experience given the most uniformly good results.

I am not yet convinced of the wisdom or need of sacrificing a normally functioning turbinate and running the risk of producing annoying intranasal discomfort as a result, for the purpose of rebuilding an external depression of the bridge. I feel that this opinion is strongly supported by the fact that cartilage is so readily accessible and can be molded and handled with so much greater ease, in addition to its superiority in withstanding infection and not eventually being absorbed.

The reaction locally, following the correction of these external deformities, is negligible and promptly subsides. Curiously enough, the patients suffer practically no pain following operation and little or no discomfort. They are required to wear for a short time a properly molded splint, which must be suited to the individual case; therefore, the postoperative management is important. We feel that the rhinologist is the one best suited for this field of surgery, owing to his special knowledge of the parts involved.

Each case is a separate and distinct mechanical and artistic problem itself, and one should possess the necessary mechanical skill and appreciation of the proper anatomic ideal to be approached.

403 CARTER BUILDING.

CASE 1. DORSAL HUMP DEFORMITY.



Before Correction.



After Correction.

CASE 2. LATERAL DEFORMITY OF NOSE.



Before Correction.



After Correction.

CASE 3. LATERAL DEFORMITY OF NOSE.



Before Correction.



After Correction.

CASE 4. LATERAL DEFORMITY OF NOSE.



Before Correction.



After Correction.

CASE 5. DORSAL HUMP WITH LATERAL DEFORMITY.

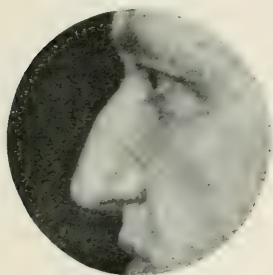


Fig. 1-A.
Before Correction.



Fig. 1-B.
After Correction.



Fig. 1-C.
Before Correction.



Fig. 1-D.
After Correction.

CASE 6. DORSAL HUMP DEFORMITY.



Before Correction.



After Correction.

CASE 7. DORSAL HUMP DEFORMITY OF NOSE WITH
DROOPING OR ELONGATED TIP.



Before Correction.



After Correction.

CASE 8. WIDE NOSTRILS OR AFRICAN TYPE DEFORMITY
OF NOSE.



Before Correction.



After Correction.

CASE 9. TRAUMATIC SADDLE DEFORMITY OF NOSE.



Before Correction.



After Correction.
By means of cartilage transplant.

CASE 10. CONGENITAL SADDLE DEFORMITY OF NOSE.

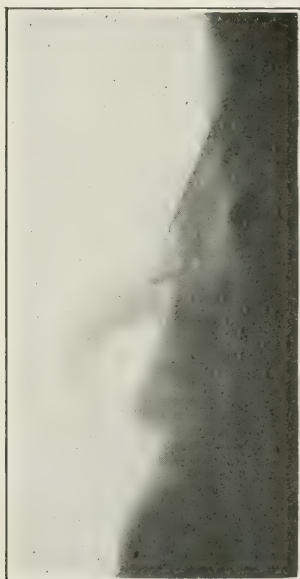
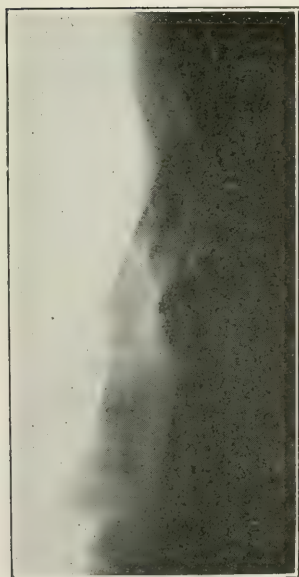


Before Correction.



After Correction.

By means of cartilage and bone
transplant.



Radiograph of Case 10 taken four months after transplantation
of bone and cartilage for correction of saddle
deformity of nose.

XLII.

A CASE OF CONGENITAL HEMIMACROGLOSSIA, WITH DISTURBANCE OF THE LOCOMOTOR APPARATUS OF THE SIDE OPPOSITE THE LINGUAL LESION.*

By J. N. ROY, M. D.,

ASSOCIATE PROFESSOR AT THE UNIVERSITY OF MONTREAL;

LAUREATE OF THE ACADEMY OF MEDICINE OF FRANCE,

MONTREAL.

After having made numerous bibliographic researches, in several languages, on the subject of which I am about to treat in this communication, I believe that I may say the following observation is unique of its kind. If we know that acquired macroglossia manifests itself as the result of inflammatory lesions of the tongue—diathetic or others—it is not the same with regard to congenital macroglossia, the cause of which has always been unknown to us. My patient was very interesting from several points of view, in the sense that the case was one of congenital hemimacro glossia, accompanied by hypertrophy of the corresponding cheek and troubles of the locomotor apparatus of the side opposite the lingual lesion. This association of symptoms particularly attracted my attention up to the point of inducing me to advance a hypothesis to explain by a unique lesion all the phenomena observed. Here is the history of my little patient.

Case Report.—On the 26th of November, 1919, Mrs. C. L., who came from Gaspé, asked me to make an examination of her daughter, three and a half years of age, for a lingual affection dating back to childbirth. She informed me that at that time her baby weighed twelve and a half pounds, and that although the doctor was not obliged to apply the forceps or to practice version, the confinement was very severe. The cord, very voluminous, was long and rolled round the neck. The face was cyanosed, and the tongue, blackish in color, hung

*Read before the 10th International Congress of Otology, Paris, July, 1922.

out of the mouth. In order to reanimate the infant the doctor had to resort to artificial respiration for several minutes. The left cheek was more developed than the right, and it fell lightly on the corresponding lower jaw. There existed certain phenomena of atrophy and paralysis of the upper and lower limbs of the right side. During the five following weeks the tongue slowly diminished in size and recovered its normal color. However, always very large, it protruded from the lips for about two centimeters, and after a lapse of time that condition seemed to remain stationary. The left cheek also became smaller by degrees, but remained projected a little more than the right. The child took the breast with some difficulty.

Six weeks after the birth of the child the parents went to consult Dr. Mireault, orthopedist of Montreal, who was at that time holding his summer clinic on the shores of the Gulf of St. Lawrence, and sought advice in relation to their infant's hemiplegic trouble. My excellent confrère, who subsequently sent his little patient to me for the lingual treatment, was good enough to furnish me with the following notes in relation to his specialty:

"At the examination I noticed that the upper limb and the lower limb of the right side were smaller and shorter than those of the opposite side, which were normal. The arm rested inert along the body, and the leg was without movement. The tissues lacked firmness in the paralyzed regions.

"In that part of Canada nature offers the greatest advantages for the restoration of health, but civilization has not yet carried its progress there; hence we have not yet the assistance of electricity there, either for diagnosis or for treatment.

"The doctor who assisted at the confinement, and whom I met a few days after my first examination, declared to me that he did not find granulations in the membranes or placental tissue.

"The father and mother, who were both about thirty years of age, are in excellent health and do not indulge in alcoholic liquors.

"There does not seem to be either syphilis or tuberculosis in their family.

"They have two other children, both in good health, and whose confinements were normal.

"In face of these symptoms, once a week I carried on massage, accompanied by passive movements of the paralyzed limbs. This treatment was practiced for three months, and at the end of that period a slight mobility manifested itself. The following year I noticed a progressive amelioration. The child could now move the arm and leg. However, these limbs had not yet attained the vigor of those on the opposite side. During this season, as well as during the two following summers, massage and passive movements were continued. I noticed a gradual diminution of the troubles of the locomotor apparatus to the right. The child commenced to walk at the age of eighteen months, and when three and a half years old could run and jump. However, her walking is a little defective, for there is a slight lameness. During the whole period of her orthopedic treatment I never observed spasms or contractions, and the articular movements were executed in their normal amplitude and without pain. There always existed a slight shortening of the right arm and leg, as well as a certain degree of atrophy. These phenomena, with a little natural feebleness of the arm, will be permanent in the future. The tongue and cheek are the only two parts whose condition seems to have remained stationary during the last three years."

At my examination I ascertained, in addition to the symptoms already described, that this girl, having always breathed the pure and saline air of the Gulf of the St. Lawrence, is perfectly developed for her age. The skull, of ordinary size, is well formed and the child's intelligence is above the average.

Her mother informed me that from time to time the child had sudden changes of humor, and even violent fits of temper. However, these cerebral phenomena are becoming less and less frequent. She has never had convulsions.

The left cheek is a little larger than the right, at the same time being very firm.

There is no facial paralysis.

The pupils are normal and there is no nystagmus.

The hearing is excellent on both sides.

The tongue, very thick on the left side, is deviated to the right and constantly hangs out of the dental arcades to a length of nearly two centimeters. Its hypertrophy is uniquely localized on the left side and continues to the base. Of normal

color, it is a little rough to the left, and presents on that side a firmer consistence than on the other. There are no sublingual varicose veins, and the teeth have not produced any ulceration.

The inferior lip is lowered without being increased in size.

The teeth are well implanted, and the gums show nothing out of the ordinary.

The submaxillary glands are not hypertrophied.

The parents have never attempted to force the tongue back into the child's mouth and hold it there by means of a bandage. Moreover, that organ could not be contained in the buccal cavity.

Saliva oozes from the mouth slowly.

There is no paralysis of the muscles of the tongue, and the taste seems to be normal on both sides.

No pain has ever been felt.

The tonsils have not increased in size, and nasal respiration is excellent.

The roof of the mouth is normal.

Mastication is strongly impeded, but swallowing is accomplished relatively well.

Phonation is a little troublesome.

In the presence of this congenital hemimacroglossia, which seemed to have remained in a stationary condition for three years, I immediately proposed the ablation of a part of the hypertrophy as being the most rational treatment. The accepted intervention was fixed for November 29.

Operation.—The little patient was put under the influence of chloroform, and I immobilized the dental arches with a mouth gag. Two tongue forceps were afterwards applied to the end of the tongue—one of them being held by an assistant—and after having practiced a light traction, I divided the organ by means of a pair of scissors for the length of about five centimeters in following the middle raphe to the left. Once the tongue was separated in two, the affected side being longer than the healthy side, I removed a V shape piece in order to reestablish the symmetry. The section commenced on the left external part, at a certain distance from the tip, and continued towards the base on a line of four centimeters. After having controlled the hemorrhage, which was insignificant, I made a series of sutures commencing at the wound on

the floor of the mouth. When all the points were placed I applied a last transversal suture having the form of a U to properly coapt the middle part. At this time the tongue entered the mouth perfectly, and its tip, which was drawn neither to one side nor to the other, maintained its regular anatomic design.

As a buccal antiseptic I prescribed a solution of resorcin to be used with an atomizer.

The outcome of the operation was normal, and after several days of lingual reaction the edema disappeared gradually. I commenced to make the ablation of the sutures at the end of the first week, and on the eleventh day they were all removed. Three weeks after the intervention the girl, thoroughly well, returned home with her mother. The left side of the tongue was still a little larger than the right; however, its volume diminished little by little.

The anatomicopathologic examination of the piece that was removed showed that we were in the presence of a hemimacroglossia of lymphatic form—a microscopic variety most generally observed in this kind of lesion of congenital macroglossia.

From time to time I received news of my little patient, and in June, 1922, the left cheek was of almost equal volume to that of the right cheek. The left side of the tongue was slightly larger than the right; however, it was quite mobile and easily contained in the mouth. Considering the age of the child, her pronunciation was as good as could be expected and her temper was improved.

As it is now a little more than two and a half years since the operation was performed, I have every reason to believe that there will not be any relapse of the lingual affection.

The upper and lower limbs of the right side are still slightly shorter than those of the left, and at the same time present a little atrophy. These troubles will never wholly disappear in the future.

To sum up the case, a child born of parents enjoying the best of health, without any diathesis, has at birth a hemimacroglossia to the left, accompanied by hypertrophy of the corresponding cheek, and paralytic phenomena of the locomotor apparatus on the side opposite to the lingual lesion. After

the operation the tongue and cheek returned to an almost normal condition, but the motor troubles of arm and leg, though very considerably improved, did not entirely disappear.

If now we stop to consider the cause of this strange disease, we see that in the orthopedic domain the etiologic factors of the locomotor troubles of newborn children can give rise to very different interpretations, according to their localization.

Lesions of the nervous system are more serious than articular lesions, and the contusions of the muscles do not give a prognosis as serious as in the case of epiphysary tearing.

Affections of newborn children resulting from abnormal conditions during the intrauterine life manifest themselves at birth or in the first days after confinement.

Tissues in the course of growth are very vulnerable, and their power of resistance is less during intrauterine development than on the nursing child.

Our slight knowledge of fetal life does not give us an entire comprehension of abnormal phenomena, mechanical or nutritive, which have for result a being infirm or deformed when brought into the world.

There are three classical types of congenital paralysis. The whole three affect only the arm. The first paralysis attacks the entire arm; the second, that of Klumpke, settles in the forearm; and the third, the paralysis of Erb, occupies the upper region, the latter kind being much more frequent.

But apart from the present instance, we have never yet observed a case of congenital paralysis of the lower limb.

The totally transitory paralytic character of the troubles of the patient in this case inclines me towards two etiologic factors: either a bulbar capillary hemorrhage or in the nervous trunks, or else a traumatism of the articular capsules due to a slight and prolonged traction on that membrane by an exaggerated flexion during the last days of intrauterine life. Nevertheless, I must say that the arm and leg both participated in the state of paralysis, that there was neither contractions nor bad positions of the limbs, and that symptoms of pain were absent.

As to epiphyseal tearing, it must be eliminated.

The cause of congenital macroglossia is not yet known.

In my case, I believe the hemimacroglossia can be attributed

to the same etiologic factor as the affection of the locomotor apparatus. Indeed, if we had been in the presence of a cerebral lesion, there would have been the phenomena of contraction, pain and sensory troubles. None of these were present, and, more than that, the intelligence remained lively, the character became normal and physical development followed the ordinary course of growth.

With a meningeal edema there would probably have been persistent lesions being connected with a diathesis.

Consequently, in the presence of the association of symptoms observed in the case of my little patient, I consider I found sufficient elements to justify the hypothesis of a bulbar capillary hemorrhage in the region of the nucleii of the seventh, twelfth and ninth cranial nerves on the left side, a lesion which determined a trophic trouble of the tongue and cheek, accompanied by divers troubles, transitory or permanent, of the upper and lower limbs on the side opposite the lingual affection.

716 ST. HUBERT STREET.

XLIII.

SINUSITIS FROM SWIMMING.

BY RALPH A. FENTON, A. B., M. D.,

PORTLAND, OREGON.

With the very great increase of public interest in swimming, diving and the use of public tanks and bathing places, it is often alleged that disease is spread by such resorts. Spasmodic efforts to regulate public baths have been succeeded by a rather coherent policy of water examination by health authorities, and by insistence upon preliminary showers, provision of heat sterilized suits, as well as chlorination, ultra violet ray treatment, and other bactericidal measures for the water itself.

Nevertheless the incidence of ear, nose and throat disease following swimming is still commonly noted by rhinologists and is often blamed upon dirty water. Certain fallacies in this generalization have caused us to examine statistical findings in our own city and in other fields, and to present a series of typical cases, so that free discussion of the subject might occur before this academy.

The bacterial content of public tanks in Portland runs from 30 to 4800 per cc.;¹ that of the Willamette River as it enters the city limits after traversing a populous valley for 135 miles is, in June 600 per cc.; the river water offshore from the mouth of one of the city's largest sewers contains only 2,000 per cc.² Published figures for the River Thames are from 277 (April) to 2,075 (January); for the Illinois River at Ottawa, 6,300 to 8,200 (May); and for the Mississippi at New Orleans, 805 (August) to 3,597 (April).³ The initial purity of Portland's drinking water, which plates sterile throughout the year, and is brought thirty-five miles from a closed forest reserve near Mount Hood, doubtless is responsible for the low tank figures. No sewage infection was found in tanks or river. It is of course relatively hard to grow for differentiation of the bacteria toxic to human beings, from the ordinary counting plates; for accurate identification animal experimentation is necessary, and not much work has been done in this

field. Overgrowth of these germs which are viable in cool water for long periods, not requiring warmth or serum media for favorable growth, is responsible for much of the negative information regarding human disease germs in water. But, by the same token, it is scarcely possible to conceive that human germs of highly toxic action upon the respiratory mucosa can multiply under conditions prevailing in swimming tanks, rivers or lakes. Low temperatures, unfavorable media, antiseptic agents and the action of air and sunlight upon agitated masses of water assure the subdivision and disinfection of most of these contaminations. Samples from various depths count very much the same.

J. W. Robinson⁴ mentions certain effective rules of the California State Board of Health, requiring that proper and convenient places for expectoration by bathers be provided; that surface contamination shall be skimmed off; that the water shall flow off at various levels continuously; and that all persons known or suspected to have infectious diseases shall be excluded. He asks pertinently who will do the excluding, and, in common with Walker,⁵ suggests enactment of law making it a misdemeanor for an infected person to enter a tank.

A committee of the American Public Health Association has recently reported upon a questionnaire regarding bathing places, which had been sent to 2,000 eye, ear, nose and throat specialists. Tanks were considered important means of spreading "colds" by 67 per cent of the 600 men who made detailed answers. Sinus infections were reported by 72; one man reported an epidemic of sinusitis. One hundred and forty-five cases of otitis media were reported; 4 cases of meningitis were attributed to nose or ear complications. (It is of course likely that many cases reported as simple "colds" were really acute sinus or ethmoid infections.) The whole difficult matter of protection of tank water and of testing for the germs of upper respiratory and conjunctival disease was referred to the laboratory section of the American Public Health Association for report at this year's meeting.

From our own cases we have selected four rather typical examples of infections following swimming which will be reported briefly:

1. Acute left ethmoiditis and maxillary sinusitis; latent chronic left frontal sinusitis; frontal lobe abscess; bilateral jugular thrombosis. Fatal.⁷

N. L., white boy, 19, referred by Dr. R. B. Stephenson of Centralia, Washington, who had opened his left ethmoid, removed part of the left middle turbinate, and washed out his left antrum. This had been done because of severe acute sinusitis with frontal headache, following upon several months of swimming and high diving, mostly in rivers and stagnant ponds, never in a public tank. Yellow pus flowed freely after this drainage, from the left frontal, with temporary remission of symptoms. In a few days, however, temperatures to 104 and upper lid edema pointed to orbital extension, and Dr. Stephenson brought him to Portland. Frontoorbital radiographs were inconclusive.

An extensive Luc-Hajek external frontal operation was done at once, October 13, 1920. Free nasal drainage was found, and it was assumed that a tendency to point toward the external angular process through the upper lid would cease, following the open nasal channel. Temperature went down to 99 for a day or two, but speedily rose to 104 and over, with a relatively slow pulse. The optic discs were somewhat choked, especially in the left eye, so that after negative lumbar puncture diagnosis of frontal lobe abscess was made. Cord-like swelling and sternomastoid soreness, with a white count mounting to 17,500, 87 per cent polymorphonuclear, caused the suspicion of right jugular thrombosis.

The entire inner wall of the left frontal sinus was removed October 23, 1920, by Dr. Dillehunt, opening a large epidural abscess which gushed out under pressure, accounting for the constant flow of pus into the nose through dehiscences not found at the first operation and also for the fistulization toward the outer side of the orbit.

Metastatic lung involvement was expected; the jugular soreness became bilateral. Fibrinous pleuritis and bronchopneumonia became manifest, especially on the right side. Remarkable stimulation was produced by several intramuscular injections of whole blood by Dr. Bean. The patient's mental condition remained bright at all times. Some herniation of the

frontal lobe took place, partially collapsing the half centimeter soft rubber drain through which dichloramin oil had been injected daily with almost complete cessation of the yellow discharge. A stiffer tube was used, but about three weeks after the brain abscess was drained heavy grumous bloody material escaped one morning, and it was feared that this meant an extension of infective thrombosis beyond the abscess area. Next day the right arm was paralyzed, and aphasic phenomena and semicoma a day later mapped out the march of meningeal invasion. Death came on the 27th day postoperative.

Postmortem by Dr. Menne the same evening disclosed an abscess extending vertically about 8 cm. from the left orbital roof and diagonally to the right across the longitudinal sinus, eroding the inner table of the frontal bone about 2 mm. The entire left hemisphere was compressed by a quantity of pus filling the dural cavity. Pus had burrowed through the orbital roof, traveled subperiosteally toward the outer angle, to appear under the eyebrow. There was no pus in the ethmoids, maxillary antra, sphenoids or right frontal.

Most unusual was the finding of complete and organized right lateral sigmoid and jugular thrombosis and incomplete organized thrombosis of the left sigmoid and jugular. There were hundreds of lung abscesses, also bilateral coalescing bronchopneumonia, marked acute hemorrhagic and fibrinous pleurisy, acute tracheobronchial lymphadenitis and marked cloudy swelling of the parenchymatous organs. The chief organism involved was a hemolytic streptococcus.

2. Acute left ethmoiditis; orbital cellulitis; coma; radical external ethmoid operation; recovery.

A. K., white boy, 11, poorly nourished, sent in to Multnomah Hospital September 16, 1920, on account of pain and swelling over left eye following swimming in river about a week previous. Lids were swollen one day prior to admission. Temperature was 99.8, leukocytes 11,000. Acute anterior ethmoiditis was diagnosed, and as it did not improve with saline hot nasal irrigations and seemed to be pointing toward the inner end of the left upper lid, this region was incised freely, September 29, with discharge of much staphylococcic pus. The fistula came from the anterior ethmoid, and

washed readily down into the nose. Under dichloramin oil irrigation this fistula ceased to discharge pus after two weeks, and as the temperature remained normal he was taken home November 1.

Thirty-five days after the first incision, on November 5, he was brought back, very dull mentally, temperature 99, white cells 13,000, pulse 68. The ethmoid still showed dark in the radiograph. As the temperature rose slightly the pulse reached 62 and 64. Radical external ethmoid was done on the left side, November 8, opening the posterior cells freely. There was little pus in front but much in the posterior group.

The highest postoperative temperature was 100.8, next day; the semicoma disappeared at once. Diplopia persisted for ten days. This boy left the hospital well two weeks after the radical operation and is still well; he has quit swimming.

3. Acute left ethmoiditis; orbital cellulitis; double frontal sinusitis; external radical frontal and ethmoid operation; exposure of dura; recovery.

A. H., white boy, 12, robust, admitted to Multnomah Hospital December 12, 1921. This lad had suffered from pains in his face and over his eyes following diving during the preceding summer. He had been sick for two months, and a swelling over the left eye had been opened by a neighbor, four months before admission. Yellow streptococcic pus had discharged from the fistula since. Temperature was 97.8, pulse 74, white count 9,500; radiograph showed dark left frontal, with no apparent frontal involvement.

Radical external left ethmoid operation December 15, 1921, by Dr. Lupton. There was little posterior involvement, but a rather large left frontal, placed almost horizontally, with no extension upward into the frontal bone, was found to communicate directly with the right frontal. The partition between was loose, coming away as a sequestrum. An area of dura 1 by 2 cm. was exposed above the left frontal.

Postoperative temperature ran from 97 to normal. There was no diplopia, and the boy left the hospital nine days postoperative and has been well since his discharge from office care a few weeks later.

4. Chronic right maxillary sinusitis; chronic bilateral ethmoiditis; chronic catarrhal otitis media. Acute exacerbations; radical antrum and internal ethmoid operations; recovery.

M. W., white woman, 32, an expert swimmer and exhibition diver for some years, had been troubled with head discharges for several months prior to the autumn of 1919; hearing grew worse after each season of diving. She liked to dive because it "cleared out her head," but she was always careful to expectorate mucous into the side gutters of the tanks. Her frontals were clear, antra and ethmoid dark. Free foul curdy pus was irrigated out of the right maxillary, which yielded to treatment only to recur promptly whenever she began diving.

Radical right antrum (Luc-Caldwell) was done January 17, 1922, with removal of much polypoid mucosa and prompt recovery. The streptococcus predominated in this infection. In spite of frequent warnings she swam a few times more, causing such ethmoid exacerbation that double Mosher ethmoidectomies were done in June, 1922.

Practical cessation of pus has already taken place; the patient has already gained fifteen pounds and hears about 10 per cent better than before the radical antrum. She does not swim now.

Such cases have abounded in the literature, but have infrequently been ascribed so clearly to swimming and diving. Cobb,⁸ in 1908, stressing the fact that those who take tub baths do not put their heads under the water, cites a boy of 15 who developed ethmoiditis after five visits to a public tank; a man of 20 with acute otitis media; and a boy of 14 with double ethmoiditis and double suppurative otitis, having tender mastoids. All recovered and remained well when they stopped swimming.

Wilkinson⁹ in 1912 and Hope¹⁰ in 1914 cited a number of cases before the Royal Society of Medicine.

Wilkinson reports a man of 19, with frontal sinusitis existing for two years following diving; a woman of 32, with a suppurating maxillary antrum for six months following a "ducking" in a public tank; a boy of 17 with chronic ethmoiditis who returned from the seashore with an "orbital abscess"—osteomyelitis of the frontal bone, both frontals filled with

pus, bilateral subdural abscesses, left frontal lobe abscess, and death two months later; and a boy of 15, delirious, with "orbital abscess"—double frontal; "pyemia of the lungs", following one day after swimming in baths, and fatal one week later. The discussion turned upon strong prohibitory measures against permitting children with nasal discharges to enter public bathing places, even at the seashore. Hope reports two frontal cases: a man of 20, swimming four days before, orbital pain and swelling the next day, found to have a maxillary antrum and an orbital abscess with fistula down from the frontal floor. Discharge recurred until a radical Killian was done, when the dura was found exposed, two months later; well after three months. His second case was a girl of 14, of similar history. Polypoid granulations pointing to chronic involvement were found on her dura where exposed. This case had no maxillary involvement. It was developed that both these cases dove feet first and was again maintained that no person with an existing nasal suppuration, however mild, should swim in a tank or pool.

The normal protective mechanism of the sinuses, as stated by Skillern,¹¹ includes (1) the ciliated epithelium, with its wavelike action toward the ostia, and (2) the inhibitory action of sinus secretion upon germ growth.

Swimmers and divers introduce the following factors which interfere with this protective apparatus:

1. Destructive action of plain water upon cells, by osmosis, drawing out saline elements, causing edema and acidosis of cells, loss of ciliary activity and eventual cell death. This "irritative action of water" causes lowered local resistance. The specific protective substance of the sinus secretion is diminished and eventually lost by being forcibly washed away and later inhibited by the edema and chemical degeneration of the secretory cells.

2. Washing into the sinus of pathogenic and saprophytic germs from the more highly immunized direct airways of the patient's own nose and throat; retention of these pathogenic germs in a warm cavity lined and often closed off by edematous cells with impaired resistance. The germs of vegetable decay found mainly in water, multiplying by preference at low temperatures and in the open air, must not be considered to

have much to do with sinusitis. Nor is it wise to assume that germs from other persons are directly to blame in all cases. Too many people swim alone or in large bodies of outdoor water and are nevertheless infected. Contamination from other swimmers directly is made less likely by the coolness of the water, its constant agitation, and the consequent high dilution of sputum found in it. The assumption that most persons succumbing to sinusitis after swimming are poisoned by their own nasopharyngeal germs, mechanically driven into these cavities under conditions favoring rapid growth and retention of secretions, has much justification.

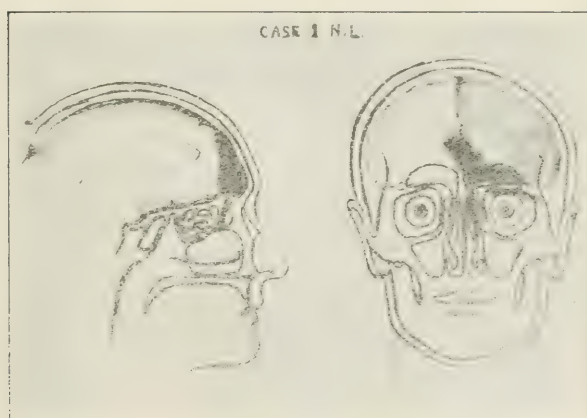
3. Direct trauma to ostia, to mucosal linings and to bony dehiscences by forcible inrush of water, especially if chlorinated, and by the forcible outrush of air bubbles. Increase of pressure in deep diving, with the bubbling and squeaking and persistent headache reported by certain swimmers, must also be reckoned with.

Prevention of sinusitis from swimming would seem to be largely a question of warning those with latent nasal infection, "chronic colds" and the like, to keep out of the water. Warnings of danger to the individual are far more impressive than warnings of danger to others; there should be an appeal to self-interest. People with high narrow noses, occlusive deflections or impinging middle turbinates are all dangerous swimming risks. Persons with nasal discharges, "colds" and "catarrh" must be warned of their own danger by strikingly worded placards. Let them stay out until they are well. Operative prophylaxis might include measures to free the drainage of all sinuses; infraction or partial resection of middle turbinates, submucous septal resections, removal of agger nasi or bullar ethmoid cells. Osteal occlusion by swollen mucosa should be made impossible. Heavy paraffin oil may be dropped in the nose before swimming. Deep diving should be forbidden to all whose sinuses are large and ostia small.

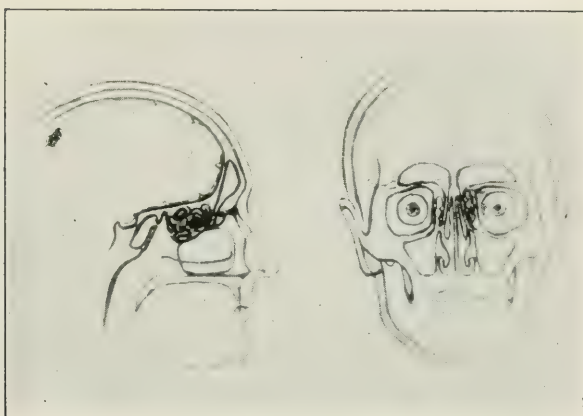
It would seem that in harmony with the work of the laboratory section of the American Public Health Association, this academy might well advise the profession and the public regarding these safeguards and dangers of diving and swimming.

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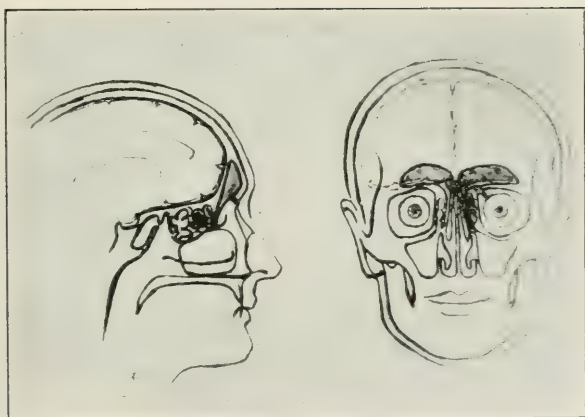
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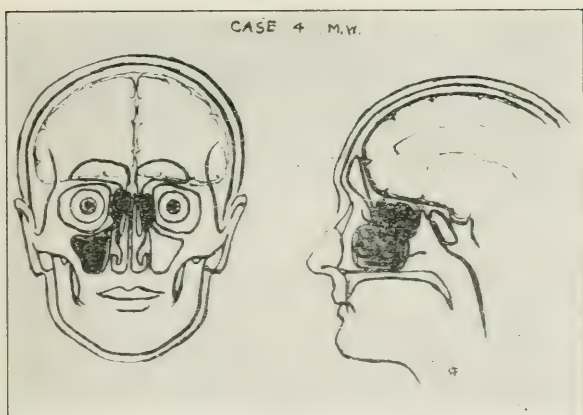
Case 1



Case 2.



Case 3.



Case 4.

XLIV.

THE EFFECT OF PRESSURE CHANGES IN THE
EXTERNAL AUDITORY CANAL ON ACUITY
OF HEARING.*

BY AUGUSTUS G. POHLMAN, M. D.,

DEPARTMENT OF ANATOMY, ST. LOUIS UNIVERSITY,

AND

FREDERICK W. KRANZ, PH. D.,

WALLACE CLEMENT SABINE LABORATORY OF ACOUSTICS,
RIVERBANK LABORATORIES, GENEVA, ILL.

The advantage of collaborative research is clearly indicated in problems connected with the sense of hearing. It is only through applying the recent advances made by the physicist to the structural conditions as established by the morphologist that the details in the mechanics of sound transmission from the drum membrane to the end organ may be properly investigated. This paper, as well as others to follow, has been made possible through the courtesy of Colonel George Fabyan of Riverbank, Geneva, Illinois. The physical material is contributed by Kranz, and the physiologic aspects, both subjective and objective, are by Pohlman.

Up to the present no satisfactory method has been devised for testing acuity of hearing in any form of animal excepting man himself. This fact necessarily places a limit on the possibility of experimental work. The most sensitive type of physical apparatus for the detection of sound is illustrated by the Webster phonometer. The moving member in this instru-

*Note.—The problem of middle ear mechanics was to have been discussed in three chapters, two of which have appeared in this journal. Since writing the first two articles the writer has associated himself with the Riverbank Laboratories, where facilities have been developed for the quantitative determination of both air and bone transmitted sound. The third paper will be deferred for this reason until the researches on the acoustic insulation of the labyrinth and the investigation of bone transmitted sound in reference to the usual otologic tests have been completed.—A. G. P.

ment is a small disc diaphragm supported in the mouth of a resonator by three tension wires. The tension of the wires and the size of the resonator are so regulated that the moving diaphragm and the resonator have the same natural period. The diaphragm is therefore very sensitive in its response to tones of this frequency, and by means of a projecting pointer the diaphragm motions are converted into torsional vibrations of a vertical tension wire upon which a small mirror is mounted. Vibrations of the diaphragm and so also of the mirror cause a change in the width of a beam of light reflected from the mirror into a ruled ocular eyepiece. The intensity of the sound, within certain limits, may be determined from the width of this band. The instrument, being carefully tuned, is useful for only one frequency.

The sound transmission mechanism of the ear, on the other hand, is an entirely different type of apparatus. It is not tuned to any particular frequency and does not possess a resonator to reinforce the different sound pulses impinging on the drum membrane. The element to be moved is not a minute mass with a definite restoring force and operating only upon another light mass, as in the mirror of the phonometer. The hinged bony apparatus of the ear is relatively heavy and not only does not present the definite restoring forces noted in the phonometer; moreover the system operates against a column of water (endoperilymph) in the inner ear.

The drum membrane presents a diaphragm which is flanked by air on both sides. Unlike the diaphragms used in the physical laboratory, this membrane is not flat in either birds or mammals. The mammal presents the typical umbo-in picture found in man, while in birds the drum membrane is convexed outward into the external auditory canal. Reptiles, like turtles, have slightly convexed drum membranes, which are composed of hyaline cartilage and directly continuous with the extra columella. The cartilage plate is always closely adherent to the overlying skin and loosely attached at its periphery to the adjacent head bones. The tailless amphibians also possess a surface drum membrane which is considerably thickened and which contains many smooth muscle fibers.

The middle ear as a moist vascular area shows the property of absorbing the contained air so long as the gas tension in this

cavity is in excess of the gas tension of the blood lymph system. There is no evidence that the drum membrane is permeable to air, and neither is it likely that the mucous membrane of the middle ear can actively secrete gases after the manner of the air bladder in fishes. Accordingly, in continuing the comparison with the phonometer, while this instrument operates with equal normal air pressure on both sides of the disc, this condition does not obtain for the drum membrane. The recent work of Fowler¹ demonstrates the presence of a slight normal negative pressure convincingly. This is in agreement with what is commonly accepted and is opposed to the contention of Secchi², who holds for a plus pressure, and of Wrightson,³ who figures the pressure on both sides of the drum membrane as equal.

It is also well known that in cases of prolonged closure of the tuba auditiva, the absorption of air from the middle ear results in retracted, hypertense drum membrane, accompanied with a conspicuous decrease in the acuity of hearing. In these cases insufflation of air into the cavity of the middle ear through the tuba auditiva not only relieves the condition of the drum membrane but also has a pronounced effect on the deafness. It is also common knowledge that some individuals suffering from tubal occlusion claim they hear better on rainy days than on bright clear days. They hear better when the barometer is low. Cases have also been reported where certain deaf people have had acuity of hearing improved, perhaps only temporarily, as a result of rapid ascent of high mountains or in altitude climbs in an aeroplane. These cases may apparently be explained as due to changes in the relative pressures in the external and middle ears.

The drum membrane in the mammal, by reason of its shape, its intrinsic elasticity and the thrust factors found in the elasticity of the incudal ligaments, has a tendency to displace lateralward in a position of absolute rest. The decrease in the depth of the umbo which often accompanies the act of swallowing may therefore not be necessarily due to the manner of the tubal closure, as some authorities maintain. The tubal orifice is opened by the contraction of the *M. tensor veli palatini* which, like the *M. tensor tympani*, is found only in mammals. These two muscles arise both phylogenetically and onto-

genetically out of the pterygoid complex and are therefore both innervated directly by motor division of the trigeminal nerve. While a functional interrelation between these two muscles has been hinted at by a number of writers, such a relation has not as yet been experimentally demonstrated. Some individuals seem to have a voluntary control of the *M. tensor tympani* without influencing the tubal orifice in the least (see cases reported by Mangold¹). If, however, a sufficient degree of negative pressure develops in the middle ear, the *M. tensor veli palatini* may be stimulated to contract and open the tuba auditiva. The *M. tensor tympani* may relax at the same time and allow a lateral displacement of the drum membrane without assuming an actual injection of the air through the manner of tubal closure. This lateral displacement probably never attains the proportions observed in forced insufflation of the middle ear cavity. The behavior of the reflex contractions of the *M. tensor tympani* are being studied at this laboratory at the present time.

The problems under consideration in this article may be stated as follows:

First—What is the optimum air pressure in the external auditory canal for acuity of hearing?

Second—What is the quantitative effect on acuity of hearing produced by increases and decreases in the air pressure of the external auditory canal?

In making these tests, a vacuum tube oscillator was used for the generation of electrical currents of the desired frequencies. The harmonics in these currents were eliminated by the use of suitable filters. A thermophone was employed for the transformation of the electrical energy into sound energy. This instrument consists of a thin strip of platinum, about 0.0002 cms. in thickness, mounted in a small case. The variations in the heating of the platinum strip due to the alternations of the current cause corresponding variations in the heating and consequent expansion of the adjacent air. These periodic expansions constitute the sound source. The strip itself has no motion. The thermophone has some advantages over the usual type of telephone receiver in the simplicity of its response characteristic as a function of frequency, in the calculation of its efficiency as a sound source and in the ease

with which the resulting sound may be reduced to minimum audibility. However, a telephone receiver was used in the place of the thermophone in some of the tests.

The thermophone case was provided with two vent holes, one of which led to a water manometer for the control and the measurement of the air pressure, the other vent being connected to an ear tip sealed in the external auditory canal of the observer with cotton and collodion, making an air tight connection. The tests were conducted in an isolated room, the floors and walls of which were lined with felt to eliminate extraneous disturbing noises. The arrangements of the apparatus were such that the observer could not see the water manometer. It was also found on test that he was unable to differentiate between a plus and a minus air pressure in the external auditory canal. Thus a bias in judgment due to knowledge of the type of pressure changes made by the operator was eliminated. Unavoidable fluctuations of attention on the part of the observer were, however, of some slight importance.

Test 1.—With the pressure in the water manometer at normal, certain pitches were exhibited to the point of minimum audibility. When the column of water in the pressure arm of the U-tube was raised and lowered slowly, the following subjective observation was made: In all instances slight increases in pressure "killed" the sound, whereas when the pressure was lowered below normal, the tone was reported as "louder," until the level of about 10 cm. water minus pressure was reached. Further decreases in pressure again "killed" the sound. This was tried repeatedly and with uniform results.

Test 2.—Determinations of the quantitative effect on acuity of hearing due to changes in the air pressure of the external auditory canal were made as follows: The minimum necessary energy (E) for audition having been determined at normal air pressure, this energy was multiplied by a factor of 4, and then the air pressure was varied until the sound became inaudible, both positive and negative pressures being tried. This was repeated with a factor of 16 and in one case with a factor of 64. These tests were made with five different frequencies: 2048, 1024, 512, 256, and 128 p. p. s. In some cases the available range of pressure variation was not sufficient to kill the

sound completely, and in the following table these are noted as "faint."

Frequency	Energy	Plus Pressure	Minus Pressure
2048	4 E	10 cm. inaudible	21 cm. inaudible
	16 E	10 cm. inaudible	27 cm. inaudible
	64 E	20 cm. inaudible	27 cm. faint
1024	4 E	9 cm. inaudible	20 cm. inaudible
	16 E	22 cm. inaudible	26 cm. faint
512	4 E	10 cm. inaudible	19 cm. inaudible
	16 E	22 cm. inaudible	25 cm. faint
256	4 E	8 cm. inaudible	16 cm. inaudible
	16 E	21 cm. inaudible	26 cm. faint
128	4 E	18 cm. inaudible	20 cm. inaudible
	16 E	23 cm. faint	25 cm. faint

Test 3.—In this experiment the minimum necessary energy for audibility was determined for three different values of air pressure in the external auditory canal—i. e., normal air pressure, 10 cm. of water plus pressure, and 10 cm. of water negative pressure. The purpose of this test was to get a quantitative measure of the effect found in Test 1. Several determinations were made at each pressure value, these being taken in random order. Two different tones were used. The average results showed that for a tone of 2048 p. p. s., the necessary energy was 0.33 E when using the negative pressure, and was 1.9 E when using the plus pressure. For the tone of 320 p. p. s., 0.7 E was obtained with negative pressure and 2.5 E with the plus pressure, the E being as above, the minimum energy at normal air pressure. The determinations of minimum audibility were more difficult at the lower frequency than at the higher.

Test 4.—Test 3 was repeated, using a small head type of telephone receiver instead of a thermophone. The receiver was sealed to the external ear with cotton and collodion and provided with a side vent for connection with the water manometer. But one was used, 2048 p. p. s., and it was found that the necessary energy was 0.3 E with the negative pressure and 1.75 E with the positive pressure of 10 cm. of water. The check with Test 3 is good.

DISCUSSION.

It is at once obvious that where we are dealing with sound the influence of the variations in the pressure of the air contained within the apparatus must be considered. The normal pressure may be taken at 76 cm. Hg., or roughly 1030 cm. of water. A change of 10 cm. will therefore only involve a factor of about 1 per cent, which is negligible. A change in pressure also involves a slight change in the volume of the enclosed cavity. However, a decrease in pressure and the consequent small increase in volume both tend to make less effective a given electrical current in the receiver. This, however, operates in the opposite direction to the effect found of an increase in acuity with a small decrease in pressure and consequently cannot serve to explain it. It should also be noted that a pressure change of 10 cm. of water is not very considerable. It is equivalent to raising or lowering an individual suddenly through a distance of about 250 feet. Greater pressures will be studied later, but other problems being considered and the number of ears available at the time was limited to two.

The effect of a positive air pressure in the external auditory canal is to force the drum membrane toward the middle ear cavity. This increases the tension of the drum membrane, but because the direction of the displacement is parallel to the pull of the M. tensor tympani the connective tissue which makes up the connection between the drum membrane and the manubrium is somewhat relaxed. Inversely the negative air pressure in the external auditory canal will decrease the tension of the drum membrane and increase the tension in the connective tissue attachments to the malleus. Apparently when air is absorbed from the cavity of the middle ear the drum membrane becomes hypertense, the connective tissue attachments to the malleus are relaxed and the pressure on both sides of the drum membrane is not equal. Perhaps all of these factors tend to interfere with the transmission of sound through the middle ear complex. When therefore the barometer is suddenly lowered by an amount corresponding to 6 or 7 mm. Hg. the pressure on the two sides of the drum membrane appears to be equalized, and the acuity of hearing is consequently enhanced. The average normal individual might not necessarily

experience any increased efficiency in hearing, because the differences are not great and the tubal adjustment may readily compensate for the pressure difference. However, in the cases of individuals with tubal closure even this small pressure variation may have a pronounced effect and the pressure adjustment through the tuba would be wanting, therefore the effect would be more prolonged. Thus it appears that the statement of certain people that they hear better on some days than on others may readily be explained.

There is also a rather common statement which may be considered at this time. It is said that a sound is heard more intensely when the mouth is held open. If the opening of the mouth causes a contraction of the *M. tensor veli palatini* and therefore a readjustment of the pressure of the middle ear, this may also be explained. However, the explanation that the sound pulses pass up the opened tuba and reinforce those striking against the drum membrane from without cannot be considered seriously. For this to occur, the two sets of sound pulses must be in opposite phases. In other words, a rarefaction phase in the one must coincide with a condensation phase in the other. The difference in the distances traveled from the source to the external surface of the drum membrane would have to be an odd number of half wave lengths. If therefore the distance from the source to the external surface of the drum membrane differed from that to the internal surface of the drum membrane by as much as five centimeters then the longest reinforced wave length would be 10 centimeters, and correspondingly the lowest reinforced pitch would be in the neighborhood of 3300 p. p. s. The difference in distance is probably always less than this amount, and therefore only very high pitched notes might be reinforced by an open tuba auditiva.

CONCLUSIONS.

The single case investigated seems to warrant the following statements:

First—The human middle ear under normal conditions is probably under a slight negative pressure.

Second—A negative pressure of 10 cm. of water in the air of the external auditory canal increased the acuity of hearing by a factor of three.

Third—A negative pressure of about 10 cm. of water appears to enhance the transmission of sound through the middle ear in three probable ways: A decrease in the tension of the drum membrane; an increase in the tension of the connective tissue attachment of drum membrane to the malleus; and perhaps a balancing of the air pressure on the two sides of the membrane.

Fourth—The more or less constant negative pressure reading seems to point to the possibility that the tuba auditiva does not open to balance the pressure on the two sides of the membrane as often as is commonly assumed.

Fifth—The variations in the acuity of hearing under these test conditions seems to indicate that steps in the intensity ratio of 5, or even perhaps 10, are sufficiently accurate for all usual purposes in determining the curve of minimum audibility.

Sixth—We submit herewith an accurate method for testing the effects of plus and minus pressures in the air of the external auditory canal which may have a distinct place in the otologic practice. It is our intention to carry on these investigations both with normal and abnormal ears. We hope to elaborate a simple method for gaining more definite information on the functional relation of the drum membrane and the malleus.

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XLV.

IDIOPATHIC PERFORATION OF THE NASAL SEPTUM. AUTOPLASTY WITH A PEDUNCULATED FLAP OF MUCOUS MEMBRANE. CURE.*

BY J. N. ROY, M. D.,

ASSOCIATE PROFESSOR AT THE UNIVERSITY OF MONTREAL.

In this short paper, I desire to draw attention to the treatment of several varieties of perforation of the nasal septum and relate at the same time the history of a patient whom I cured of that affection by means of a pedunculated flap of mucous membrane. It is known that the loss of substance of the septum occurs differently according as it manifests itself as the result of a specific or tubercular lesion (*lupus*), a cancerous lesion—which, in most cases, ends fatally—as operative lesion, accidental or deliberate, an infected traumatic lesion, a leprous or diphtheric lesion—to mention only the principal microbic diseases susceptible of producing this complaint—or even an idiopathic lesion.

The objective symptoms depend very much upon the size and the cause of the perforation and are especially characterized by occasional epistaxis, by the formation of scabs, and sometimes, when the loss of substance is small, by a nasal hissing sound. To these symptoms there are added from time to time slight pains in the head.

Generally the patients do not seek special treatment for this condition, or, if they do, they content themselves with applying an antiseptic nasal salve so long as they are not inconvenienced by this particular hissing noise. However, it must be admitted that we are powerless if the perforation is considerable, for in that case no autoplasmic method can give any result. Fortunately, however, this kind of perforation never produces any hissing noise.

If, on the contrary, the loss of cartilaginous substance is relatively small, and when the hissing, which is the motive of

*Read before the Congrès français d'oto-rhino-laryngologie, Paris, July, 1922.

the intervention, exists, it is possible to close it by means of a pedunculated flap of the nasal mucous membrane, guided by the following technic:*. In the case where the perforation is low enough to make it possible to take a flap appropriated from the upper part of the nostril, it is necessary, after application of a strong solution of cocain and a submucous injection of novocain adrenalin, to make an incision parallel to the nasal bone, varying between two and three centimeters in length. This incision must touch the upper edge of the perforation and terminate near the opening of the nostril. A second rectilinear incision is made, starting from the posterior limit of the first, in a direction anterior to the floor of the nasal fossæ and passing five millimeters from the inferior edge of the loss of the cartilaginous substance. The opening of the nostril is in its turn divided at the junction of the mucous membrane and the skin, and that section, which begins at the anterior part of the nasal floor, terminates on the same plan at the extremity of the vestibule. All the mucous membrane which is behind the perforation is taken away, and that which is in front is detached and turned back in such a way that it will close the opening of the septum. In effecting this maneuver one must be very careful to preserve, at that part of the mucous membrane, an attachment to the edge of the perforation in a manner that will give to it the best nutrition possible. A last incision of about three centimeters is made at the upper limit of the nostril and parallel with the nasal bone, terminating at the vestibule. This pedunculated flap, which must be as large as circumstances will permit, is very carefully detached and lowered in order that the middle part may be opposite the perforation. Its raw surface is then in contact with the anterior piece of reverted mucous membrane, which is also denuded, serving to close the loss of cartilaginous substance, and offering the best conditions of vitality which can possibly be desired. Finally the flap is sutured with silk to the lip of the incision of the opening of the nostril, or coapted by means of small Michel clips. One must always apply a sufficiently firm dressing, for it must not be forgotten that this intervention is made on a mucous membrane which is very susceptible to hemorrhage.

*In the description of this method we are supposing the patient is seated, with head tilted backwards.

However, thanks to the submucous injection of novocain adrenalin, the operation is almost bloodless. If the perforation is found to be placed too high to make closure possible with a flap sufficiently large taken from the nasal bone, it may be taken from the lower part of the nasal fossæ, transposing the method of operation.

It must also not be forgotten that once cut, the flaps of mucous membrane slightly retract.

The dressings must be made in an aseptic manner to hasten the healing of the uncovered part of the septum and to prevent the suppuration which exposes these flaps to mortification.

This method is much simpler than the others, while offering more chances of success. Goldstein dissects first on the two sides, the circumference of the perforation, and next removes a ring of cartilage. To close the loss of substance, he cuts from the immediate neighborhood of the wound a pedunculated flap nearly circular, and slightly larger, taken from the posterior region of the nasal fossæ. Once detached and turned back, the flap is sutured.

Goldsmith and Shurly employ with a technic somewhat different a lamella of cartilage to close the perforation and recover it afterwards with a flap taken from the septum. I should hesitate to employ their method, owing to the danger of infection and its consequences, and also on account of the difficulty of its operative procedure.

Chevalier Jackson prefers to use a piece of the mucous membrane from the lower turbinated bone.

Hazeltine makes an elliptic incision in an anteroposterior direction, circumscribing the loss of substance. After largely detaching the mucous membrane from the upper part of the nostril he makes another incision near the nasal bone, and when the double stemmed flap is lowered he sutures the lower lip to the mucous membrane at the inferior edge of the elliptic section. As with this method the perforation is only two-thirds closed, he repeats the same technic on the other side, taking this time a little narrower flap near the floor of the nasal fossæ which, being elevated, is sutured in its turn.

One of the most arduous phases of these proceedings consists in the application of the sutures, for we all know how difficult it is, by natural ways, to maneuver in the middle part

or even in the anterior third of the nasal fossæ with a needle or the small Michel clips.

With my method, coaptation of the flap is effected quite simply at the opening of the nostril, and all my incisions are rectilineal. Moreover, the phase of the operation which consists in turning back the mucous membrane of the anterior part of the perforation, to close it up, gives more stability to the flap which is applied on a denuded surface and thus increases its vitality—something which is not possible in the other processes. The method which I have described overcomes nasal hissing and the other symptoms, as the following observation shows:

Miss C. M., seventeen years of age, came to see me on February 7, 1922, in regard to an affection of the nose which troubled her very much. She informed me that three years previously she underwent with successful removal of the tonsils and adenoid growths. In March, 1921, she observed a slight tenderness of the nasal septum, which has been gradually increasing. From time to time there were epistaxis and a little cephalalgia, localized more particularly at the root of the nose. Apart from headaches she did not suffer from neuralgia. Since the beginning of her affection there was a slight nasal discharge which later became mucopurulent. When blowing the nose there sometimes appeared small scabs from the left side. In spite of the hypersecretion and the scabs the nostrils were hardly obstructed. The sense of smell was always preserved. With the aid of a mirror, the patient, in April, noticed the presence of a small reddish pimple on the left side of the septum. On the right side the nasal fossa appeared to her as being normal. This growth was accompanied by a little more local reaction, and after having disappeared in the course of the month of May, an ulceration was noticed in the same place. In the first days of July a perforation of the nasal septum appeared. From that moment the inflammatory symptoms, which were never very marked, ceased. No treatment was undertaken during the course of that affection. Although the patient's temperature was not taken, it would seem, according to information furnished, that there had been no fever. The patient acknowledged having always indulged in the habit of inserting her fingers in the nostrils. As soon as

the perforation was established she remarked a hissing noise in her nose when she breathed rather heavily or when she sang. The noise was particularly pronounced during her sleep. As she was mortified by this condition she asked me, with a certain amount of anxiety, if I could cure her.

On making the examination of the nostrils, I observed a small perforation of the nasal septum with its seat in the immediate neighborhood above the vascular area. The perforation, which was round, measured about 5 mm. in diameter. Its contours were perfectly clear, sharp and smooth. On its left side it was surrounded by a zone of very thin cicatricial tissue, a vestige of the old inflammation. The degenerated mucous membrane was symmetrically depressed in a circular manner and much more as one approached the loss of substance. The septum was very slightly deviated to the right, and the turbinated bones were in a normal condition.

On the right side the mucous membrane had undergone no fibrous transformation, and apart from the perforation this nostril was also normal.

The sensibility of the pituitary membrane was preserved. When the patient breathed rather hard a fairly considerable hissing was heard. The exterior of the nose was rectilinear.

There was nothing interesting to note in the pharynx and the cavum.

The lacrimal ducts were normal and the patient never had epiphora.

Brought up in the country, she has rather a robust constitution, and presents no symptoms of syphilis or tuberculosis.

Moreover, the whole of her family is in excellent health, and there is no disposition to rheumatism or nervousness.

In presence of this idiopathic perforation dating back for seven months, I proposed, in order to get rid of the nasal noise, to close the opening of the septum by means of a pedunculated flap of mucous membrane. This intervention being accepted, it was postponed until February 10, and in the meantime the patient returned home with a boric menthol salve for her nose.

Operation.—Though the left nostril was somewhat larger than the right and afforded more facility for my operative maneuvers, I nevertheless decided to intervene on the other

side, because of the zone of cicatricial tissue adjacent to the perforation. As a matter of fact, the ill nourished fibrous tissue would have exposed the flap to mortification or, having removed it, I might have had a piece of mucous membrane too narrow to close the cavity properly. So after having applied a solution of cocain, I made a submucous injection of novocain adrenalin in the right nasal fossa. After an interval of several minutes I made an incision parallel to the nasal bone about 25 mm. in length, touching on its course the upper limit of the perforation and terminating near the opening of the nostril. I then made another rectilinear incision which commenced at the posterior termination of the first and which, extending to the anterior part of the floor of the nasal fossæ, passed 5 mm. underneath the lower edge of the perforation. From there the mucous membrane at the summit of the triangle was totally removed. The opening of the nostril was in its turn incised near the union of the mucous membrane and the skin, and the incision continued up to the upper extremity of the vestibule. The mucous membrane was detached with very great care, as far as the neighborhood of the anterior limit of the perforation, and then turned back in a manner to close the loss of substance of the septum. Finally a last incision, 3 cm. in length, was made as high as possible and parallel with the nasal bone. This flap, which was about a centimeter wide, possessed a good pedicle and, once detached and lowered, it amply covered the part of the perforation already filled up. After having fully verified its position, I sutured it with silk to the edge of the incision made at the opening of the nostril. Although the hemorrhage was very slight, I nevertheless applied a firm dressing. This dressing was renewed at the end of forty-eight hours, and every day afterwards during the first week. The sutures were taken away eight days after the operation, and healing continued normally. The flap adhered well to the place where it was applied and covered entirely the perforation. The patient was sufficiently cured to be able to return home twelve days after the intervention. She took with her mentholated oil to continue sterilization of the nasal fossæ. At that moment, the mucous membrane, which I had turned back to fill the opening of the septum, was hardly perceptible to an anterior rhinoscopy of the left nostril.

I saw my patient from time to time, and at the beginning of May she was perfectly cured. The flap, which exceeds the limits of the perforation, is very solid. The rest of the septum is thoroughly healed, and there is no more secretion in the nose. Respiration is excellent on both sides. The nasal hissing has naturally stopped, as well as all other symptoms of which the patient complained.*

The case which I have just related proves once more to what point a simple lesion caused by a scratch can have serious consequences. Indeed, this young girl, who had a habit of inserting her fingers into her nose, one day produced with one of the finger nails an injury on the left side of the septum. A little submucous hemorrhage occurred, which was soon followed by infection, and there resulted a perforated idiopathic ulcer well described by Hajek. Happily for the girl, the loss of substance was very small, and a cure took place even without treatment. Immediately after cicatrization the scabs and epistaxis definitely ceased, and of the inflammation there remained only the nasal hissing.

According to the anterior rhinoscopy, the phenomenon can be explained in the following manner: As there had been a slight deviation of the septum to the right, and as on the left side the perforation was surrounded by a very sharp fibrous tissue, the amount of air on that side was greater on respiration than on the other, causing a suction, so that the air of the right nostril, passing through the opening of the septum, made the hissing noise. I further believe that the cicatricial tissue also played a certain part in the production of the hissing.

716 ST. HUBERT STREET.

*This patient was presented at the Montreal Medical Society in March, 1922.

XLVI.

OTITIS MEDIA, MASTOIDITIS AND DISEASE OF
NASAL ACCESSORY SINUSES AS CAUSATIVE
FACTOR IN MALNUTRITION IN
CHILDREN.*

By T. H. ODENEAL, M. D.,

OPHTHALMOLOGIST AND OTOLARYNGOLOGIST TO THE BEVERLY
HOSPITAL AND MASSACHUSETTS STATE INFIRMARY,

BEVERLY, MASS.

This report comprises the colored races only, malnutrition among the Caucasians being a rarity on the Canal Zone, where these cases were collected.

In looking up the subject of malnutrition in the textbooks on children's diseases I was enabled to find only twice mention made of the connection of the ears and malnutrition, its connection with acute otitis media only being mentioned and that in a few lines.

Of the cases of malnutrition admitted to the Ancon Hospital, Panama Canal, approximately 75 per cent had ear disease at some time during the course of the disease. These cases were admitted as a rule late in the disease, and many were hopeless at the start. A few cases came in with suppurating ears and noses, but the majority gave a negative history as to sinus or ear disease.

These little patients were sent to the eye and ear clinic as a matter of routine. A few presented the characteristic acute otitis media with relief of the high temperature and improved general condition on puncture of the drum, but the majority had to all appearances perfectly normal ear drums with the exception of a lack of luster and slight thickening, which did not prevent the light reflex.

After paracentesis of these apparently normal ear drums an improvement in the patient's general condition took place not-

*Read before the Canal Zone Medical Society, January, 1922.

withstanding the fact that the ears would not discharge for two or three days after paracentesis as a rule. The ears discharged a few days and stopped only to fill up again and rupture at intervals.

It was thought that cauterizing the lips of the wound in the drum would give more permanent drainage, and this was done with pure trichloroacetic acid, resulting in a permanent opening but without any lasting benefit to the patient, proving conclusively that not the ears but the mastoids were the foci of infection.

This delayed discharge from the ears after puncture I attributed at first to infection from the canal of the ear, but from my findings at operation on the mastoids in these cases and their behavior after operation I am now convinced the late discharge is due to slow drainage from the mastoid cavity.

The infected ears which have not been punctured drain through the eustachian tubes, the mastoid cavities filling up with pus and discharging into the ears and thus through the eustachian tubes by aspiration through negative pressure in the nasopharynx created in the act of crying, coughing, etc.

This cycle of the mastoid cavity (the mastoid at this age is chiefly antrum), filling up and discharging periodically through the eustachian tube, is repeated again and again until resolution or death occurs.

At autopsy Dr. Clark, our pathologist, found that approximately 80 per cent of the malnutrition cases were affected with mastoid disease.

Of course the question is asked whether this mastoid involvement is not a secondary condition, and this is a very vital question indeed, for on its answer hangs the lives of hundreds of infants. Before convincing myself, I was just as sure it was a secondary process due to the lowered resistance of the patient, incident to the malnutrition, as I am now of its primary origin.

Every one of these patients in which mastoid operation is called for die, without any exception, and, taking this into consideration, together with the high percentage of mastoid involvement at autopsy and the fact that although temporary improvement is obtained by paracentesis no permanent

benefit results, the probability of there being a primary focus of the malnutrition in the mastoid was to my mind excellent.

Mastoidectomy therefore was decided on all cases with ear disease that continued to decline. In all, seven cases were operated with four deaths, three of which were hopeless at the time of operation, the power of resistance being too low to permit recuperation.

The three that recovered were not expected to live before operation, and their recovery I lay to the fact that their illness was of shorter duration and their resistive power had not been undermined to such an extent that they could not come back.

The operations were performed under chloroform anesthesia and lasted from fifteen to twenty minutes, the patients showing no signs of shock.

A simple mastoidectomy was performed on the first few cases, but granulations formed so quickly and were so numerous, occasioning bleeding at each dressing, disastrous to the patient, that a modified radical was done on the later cases and found to be much more preferable. Adrenalin was injected in the perimastoideal tissues previous to operation to prevent loss of blood. After one week no dressings were required and simple irrigation sufficed until healed.

As 75 per cent of our cases had clinical ear disturbance and at autopsy 80 per cent mastoid involvement, it is readily understood what an important part the ear plays in this sad disease.

The vegetative nervous system is so easily deranged in children that the slightest irritation will cause disturbance of the metabolic function. This we see particularly while they are cutting their teeth, a rash perhaps appearing on the body, the glands becoming enlarged and vomiting of meals even occurring with rise in temperature at times.

If the teeth will cause all this disturbance while erupting, is it any wonder then the ears will do the same when attacked by an infectious process?

It is very often difficult to discover that the ears are at fault, as an abscess will form in the ears without occasioning any rise in temperature. For this reason a fresh disturbance in the patient's condition (vomiting of feedings, diarrhea, etc.) will be charged to the milk or anything but the ears, as the

attending physician naturally expects a rise in temperature when the ear becomes involved.

This is the real reason why the ears escape detection and are not looked on as vitally important to the treatment of malnutrition cases. The ear or ears will become infected, an abscess forms and discharges through the eustachian tube or drum without occasioning any rise in temperature. If the ear discharge through the drum it is detected; if through the eustachian tube it is not detected, and if the mastoid cavity becomes involved the patient slowly but surely declines.

Every case should be operated upon that continues to decline where there is a discharge from the ear or ears, everything else being negative of course.

Failure is very rapid, and just as soon as it is determined that change in diet is not beneficial operation should be done without delay.

Our pathologist, who makes it a custom to open the ears and sinuses in all autopsies, reports 50 per cent of the malnutrition cases as having maxillary sinus suppuration, often under tension. This fact alone is very important, as it affects the progress of the disease and might prevent recovery when other causes are removed.

The only solution to this I can see is to puncture every antrum in the cases that continue to decline and where pus is encountered put in a metal drainage tube, irrigating the antrum until the discharge ceases. If the antrum is irrigated by means of a canula much blood is lost at each irrigation, as the little patients bleed easily and freely, more harm than good is done, as every drop of blood is precious to them in their debilitated state.

In the patients old enough to have ethmoids these are affected quite often, but the cells are very small and will care for themselves if other causes are removed.

The findings at autopsy are based on several hundred cases from birth to three years of age.

Although skeptical at first as to the primary nature of the mastoid involvement, this series of cases operated upon, small in number, has yet convinced me that it is primary and that

many deaths are charged up to malnutrition which in reality should be laid to the mastoids and ears.

As explained before, we received our cases late in the disease, which consequently gave a high mortality rate.

Even if the history could be relied on, the greater majority of these cases could be saved, and if the patients were received into the hospital early in the course of the disease all could be saved that have mastoid involvement.

Irrigation of the maxillary sinuses in every case operated upon for mastoid disease is recommended to determine if they are affected, and, if so, treatment as recommended instituted.

CASES.

In all, ten cases are reported, seven of which were operative.

The blood, urine and feces were negative; von Pirquet positive in one.

All those operated upon were marked cases of malnutrition, with vomiting of feedings, diarrhea, slight or no temperature, and loss of weight.

Case 1 was of an infant of four months, which vomited all feedings, had severe diarrhea and was rapidly losing weight. Both drums of ears normal on admission. After a few days in the hospital the patient began to pick up and gain in weight, with finally cessation of vomiting and diarrhea.

Three weeks after admission the patient began to vomit and have diarrhea again, which continued to increase, with loss of weight until the patient was in a serious condition. Eleven days afterwards the right ear began to discharge and the patient improved, vomiting and diarrhea ceased and the patient was discharged well with a gain of two pounds over the weight at admission.

The gain in weight with disappearance of the other symptoms shortly after admission was without doubt due to proper food. The setback with vomiting and diarrhea was due to a suppurative process in the ear which on evacuation relieved these symptoms almost immediately. No rise in temperature registered the ear condition, and it was overlooked, as the ears were reported normal several times on previous examinations. This formation of abscess of the middle ear, with or without

rupture of the drum, without any effect on the temperature, is very frequent, and is the chief reason why the ear is not suspected in malnutrition cases. If this ear had discharged through the eustachian tube instead of the drum, the cause of the setback would have been laid to the feedings. Perhaps the cause of some of these ears discharging through the drum while others discharge through the eustachian tube is that the eustachian tube becomes blocked by inflammation of the nasopharynx (nasopharyngitis, swollen adenoids, etc.), causing the secretions to distend the middle ear and rupture the drum in place of discharging through the tube. This case is cited as one of many to show the importance of frequent ear examinations in malnutrition.

Case 2.—An infant of one year and five months, with marked symptoms of malnutrition. Gave history of right ear discharging for two weeks.

The patient could not retain feedings and had profuse diarrhea, with loss in weight. On admission right ear was discharging; the left ear normal, which was punctured and cauterized nevertheless with escape of a little thick mucus.

Both ears discharged next day and continued to discharge, the vomiting and diarrhea becoming worse and the patient lost weight rapidly.

Two weeks after admission a double mastoidectomy was performed, under chloroform anesthesia lasting thirty minutes. Both mastoids were filled with pus; culture from the right gave nonhemolytic streptococcus; from the left, *B. pyocyaneus*. Improvement after operation was remarkable, all vomiting and diarrhea ceasing, with a gain of one pound and four ounces in weight in eleven days. Sixteen days after operation vomiting and diarrhea began again. This continued with loss of weight up to time of death, one month and three days after operation.

At autopsy no lesions were found outside of the ears and mastoids.

Operation certainly improved this case and the patient would have lived if, to my mind, the drainage from the mastoids could have been kept free; but the granulations were so exuberant and formed so quickly, requiring constant attention, that the drainage was interfered with, causing constant ab-

sorption. A modified radical operation, which was done on the later cases, would, I believe, have saved the patient if done in the first place. The mode of onset of the illness indicated the ears were the primary source of the trouble.

Case 3.—Aged one year, admitted on the seriously ill list, in very undernourished condition, vomiting all feedings, and diarrhea.

Right ear on admission discharged a bloody serum; left drum appeared normal, but was freely incised. Both ears discharged and condition grew worse until, five days after admission, a double mastoidectomy was performed under chloroform, lasting fifteen minutes, although medical service advised against it, as the infant was certain to die, whether operated upon or not. Cultures from mastoids gave streptococcus irridans, staphylococcus albus and *B. pyocyaneus*.

Patient died next day after operation. At autopsy the ears, mastoids and maxillary sinuses contained pus, under tension in the sinuses; bronchitis and atelectasis, acute parenchymatous degeneration of the kidneys, heart and lungs.

Cause of death: Malnutrition with suppurative otitis media, mastoiditis and sinusitis as contributory causes.

Case 4.—Aged one year and two months, admitted with marked symptoms of malnutrition, very much wasted condition, vomiting of all feedings, diarrhea. History of discharging ears of one week's duration. Continued to decline and was placed on seriously ill list.

Thirteen days after admission a double mastoidectomy was performed, lasting twenty minutes, under chloroform. Pus was found in both mastoids; cultures positive for streptococcus hemolyticus. The patient did not improve after operation and died two weeks afterwards. At autopsy no lesions were found outside of ears and mastoids.

This case was undoubtedly due to early mastoid involvement, and the history was of little value, the infant no doubt having contracted the ear trouble sooner than the time given by the parents.

The prostration was too severe at the time of entrance to the hospital for the illness to have been of such short duration.

No bacteremia was present; the infant's resistive power was lowered too much to have been benefited by operation.

Case 5.—An infant of four months, admitted in a very much emaciated condition, vomiting of all feedings, and severe diarrhea. Right ear discharging at time of admission. Left began in a few days. Condition pointed to ears as cause of disturbance and operation advised but refused by parents.

Death several days after removal from hospital. Time in hospital, twelve days.

At autopsy the mastoids and middle ears were filled with pus containing *B. coli*. Maxillary sinus full of pus under tension.

This is another case which entered the hospital too late to carry out observation as to the cause of the condition and the benefit derived from proper feeding. By the time it is determined that the feeding is of no assistance in these cases it is too late to save them through operative means. If these cases were brought in at the beginning of the illness there would be sufficient time to trace the cause and to remedy the condition, but the parents are so ignorant and neglectful in this class of cases that they will allow the infant practically to die before entering the hospital. In this case the infection in the antri would probably have prevented recovery alone unless irrigated.

Case 6.—Aged one year, admitted in very bad condition, with marked symptoms of malnutrition and both ears discharging profusely. Failed rapidly, developing eczema of head and ears, became listless and apathetic, dying three weeks after admission.

At autopsy the ears and mastoids contained pus in abundance, as also did the maxillary sinuses; no other findings.

Probably this infant could have been saved if the history could have been relied on and the case operated upon as soon as admitted. If this had been a white child of intelligent parentage the history could have been relied on and mastoidectomy would no doubt have been performed at once.

This is a case of low grade mastoiditis causing disturbance of the gastrointestinal tract, with finally malnutrition, and it is cited along with Case 5 to impress the fact that the so-called

malnutrition cases are ear cases primarily, the malnutrition being secondary to the ear infection.

Case 7.—Aged one year and one month, placed on seriously ill list on admission. Both drums normal on admission, later the right discharged after paracentesis. The patient continued to decline and bilateral mastoidectomy was decided on and performed five days after admission. At operation both mastoids appeared normal. Patient died nine days after operation.

At autopsy the mastoids were found in normal state; the left maxillary sinus contained pus under tension.

This little patient was very anemic, the red cell count was only two million and the resistive power was not sufficient to allow recuperation after proper food was obtained.

This case was the only one where the mastoids were found uninvolved. It was a case of anemia and not a true case of malnutrition.

Case 8.—An infant of nine months, was quite ill on admission, vomited all feedings, severe diarrhea, both ears discharging. Due to rapid decline, double mastoidectomy was decided on and performed nine days after admission. Patient made rapid recovery, vomiting and diarrhea discontinued and gained markedly in weight. Was discharged well.

This infant would certainly have died without operation and surprised everyone when it recovered.

Case 9.—Aged one month, admitted with all the symptoms of malnutrition, vomiting of feedings, watery diarrhea, etc., and was losing weight rapidly. No history of ear trouble. Began to improve until one month after admission all the symptoms returned and it began to fail fast. Both ears were punctured several times with marked improvement in the symptoms, there occurring a gain in weight with cessation of vomiting and diarrhea after each puncture, but without any lasting benefit.

The patient's condition was such that it was finally placed on the seriously ill list and mastoid operation advised.

A double mastoidectomy was performed two months after admission, when it was found that both mastoids contained pus.

Recovery was remarkable; all vomiting and diarrhea ceased in a few days after operation and patient was discharged one month after operation with a gain of two pounds and thirteen ounces.

Although no history of ear involvement was obtained it is practically certain such existed. The drums were normal in appearance, but there was decided improvement in the patient after each paracentesis. This was clear evidence that the pus was in the ears and discharged through the eustachian tubes, the contained pus acting as a focus of infection and causing the irritative gastronomic symptoms.

On removal of the focus of infection prompt recovery took place.

Case 10.—An infant of eight months, admitted with marked symptoms of malnutrition and in a very much emaciated condition. No history of ear involvement. For almost a month first one ear and then the other would discharge, with temporary improvement each time, but the patient gradually declining until placed on the seriously ill list.

One month after admission a double mastoidectomy was performed, both mastoids found containing pus. Two days after operation improvement in symptoms became manifest, and eight days after operation all vomiting and diarrhea had ceased. At time of this report was gaining weight rapidly and pronounced well.

XLVII.

RELATIONS BETWEEN THE EYE AND EAR (INCLUDING THE VESTIBULAR ORGAN).

BY PROF. J. VAN DER HOEVE,
LEIDEN, HOLLAND.

As I am to speak to otolaryngologists as well as to ophthalmologists, I sought a subject in those parts of medical science which divides as well as unites the branches which we practice.

In doing so, the first thing that appeals to us is the relation between nose and accessory sinuses to the eye, because these cavities are so close to the orbit and optic nerve. The ear is so great a distance from the eye that it seems nearly impossible that direct influence is exercised from one organ on the other, and yet I for one believe that the relations between eye and ear, at least when we include the vestibular organ, are of much greater importance to mankind than those between nose and eye.

We may divide my subject into four parts:

1. Diseases and intoxications which cause eye and ear symptoms.
2. Eye diseases, which cause ear symptoms.
3. Ear affections, which cause eye symptoms.
4. Relations between the vestibular organ and the eye.

I will pass over the three former quickly so as to spend the greater amount of my time on the last and most important one.

We all know, that there are a great many diseases and intoxications which cause eye and ear symptoms. Of the latter we can for instance mention intoxications with chinin, optochin, salicyl, wood alcohol, etc.; of the former the well known ones as syphilis, especially the hereditary form, tuberculosis, scrophulosis, etc. I wish to ask your attention to some of the less well known and yet important diseases. In the first place, to the syndrome of blue sclerotics.

It is known that in 1910 Eddowes taught us in a very short publication the existence of a hereditary disease, in

which the sclera had a grey-blue or slate-blue color, in which the patient suffers from brittle bones; in other words, fractures of different bones occur on the slightest provocation, such as putting on an overcoat, falling on a feather bed, etc.

Eddowes thought that this was due to a lack of fibrous tissue.

Eddowes' communication was forgotten and later on the facts rediscovered, many authors having reported histories of a great many cases of this interesting disease without developing any new facts, until about four years ago Dr. De Kleyn of Utrecht and I found that these patients became deaf as they grew older.

About the same time this was also noted by an American named Bronson, who was then in Edinburgh.

I am convinced that the sufferers of blue sclerotics are nearly always subject to brittle bones and ear affection, but in the records you will find that every afflicted being has blue sclerotics; many, brittle bones and some only deafness.

This may be caused by the fact that when the blue sclerotics are present it is observed immediately; brittle bones may pass unobserved when no traumatisms occur to the patients and otosclerotics do not become aware of their trouble till later in life.

The cause is a maldevelopment of the mesoderm.

There are other infirmities, which may occur in those patients as arcus corneae juvenilis, sprains, weak ligaments, etc.

The patients often are small, crooked persons; the girl whose picture I show here, had about 27 fractures of different bones—e. g., of the spinal column, pelvis, legs, skull, etc. The most interesting feature is the affection of the auditory organ. Until a few years ago it was thought, that the inner ear could not be photographed with Roentgen rays in such a way that the photographs could be used for clinical purposes, but Dr. Stenvers of Utrecht taught us otherwise. You see here in a skull the inner ear made more visible by filling it with lead, the cochlea, the semicircular canals, the cavum tympani and the meatus auditivus internus are clearly visible.

The next lantern slide shows the same in a normal living person presenting the inner ear in a case of otosclerosis in

a patient with blue sclerotics; the inner ear is seen, a black mass, in which we can only find a few remains of the labyrinth.

As in those rare cases the relation of eye and ear is of some value, it is necessary when a patient comes to us with blue sclerotics to advise him not to enter a profession in which he is exposed to traumatism on account of the brittle bones, and to warn him that his hearing may get worse when he grows older. Professions in which acute hearing is a first duty, as telegraph or telephone operator, Marconi operator, etc., should not be chosen.

In the second place I wish to mention the eye affections, which often are found in congenital deaf and dumb people; as pigmental degeneration of retina, either in the form of the well known retinitis pigmentosa or in other forms.

It may be of some interest on this account, that in making ophthalmoscopic examination in the congenital deaf and dumb animal race, such as the Japanese or Chinese dancing mice, I found in every specimen examined a pigment-degeneration of the retina, sometimes in a form very much like a retinitis pigmentosa, but at other times more like degeneration after a chorioretinitis.

Though this fact is very easily ascertained by ophthalmoscope, histologic investigation is not so easy, but I hope we shall also be able to prove this fact anatomically. In any event both facts show a very intimate connection between retina pigment and deafness.

I wish to consider still a third instance of disease with eye and ear symptoms.

We know a peculiar disease under the name of tuberose sclerosis of the brain, in which as a rule idiotic epileptic young people have tumors and cysts in the brain, in the heart, the kidneys, the thyroid gland and in the skin. I have now observed in this disease tumors of the optic nerve disc and of the retina until now unknown.

There is another nerve disease, which we know under the name of the Recklinghausen disease, in which there are multiple neurofibromata in the skin and at the peripheral nerves and in the acoustic nerve.

Some neurologists, as Bielschowsky, etc., think that both

diseases are one and the same, only with different localization of the tumors, and consequently they speak about spongio-blastosis centralis, peripherica and universalis.

Nieuwenhuyse, on the contrary, is convinced that those diseases are two entirely different, well defined affections not to be associated. A couple of months ago I examined in the clinic of Prof. Winkler in Utrecht a patient, who was deaf, feeble sighted and had atrophy of many muscles. Diagnosis was made of meningitis with consequent deafness and muscle atrophy, but when Dr. Stenvers made X-ray photos of the inner ear to find out what was amiss, he discovered on both sides a considerable enlargement of the porus acusticus internus, which as you can see on these skiagrams did enlarge still more after a lapse of time.

So it seemed highly probable that the deafness was caused by a tumor of the acoustic nerve; the patient was reexamined and they found some neurofibromata on his back. The diagnosis was therefore changed to Recklinghausen's disease.

The vision of the right eye of this man had been reduced for many years caused by a retinal detachment. We find in the retina a huge mass, which looks like the remains of a Coates disease but it may be a tumor mass. There is a tumescence at the optic disc, which possibly may be a tumor of the disc. However, this eye is too much altered to permit a reliable diagnosis. The other eye shows a choked disc (5 diopters), and there are also in the retina two small tumors ophthalmoscopically just like the tumors in the cases of tuberose sclerosis of the brain.

Though the real proof can only be established by histologic examination of the eye, I am thoroughly convinced that this man has the same kind of retina tumors as patients with tuberose sclerosis show.

If this proves to be true, it is highly probable that the disease of Recklinghausen and the tuberose sclerosis are diseases at least very closely related to each other, since it would be almost inconceivable for such rare tumors to develop in different diseases. So you see how important again the eye and ear symptoms are. Ear therapy enabled me to make the diagnosis of the Recklinghausen disease and eye pathology may perhaps help to solve this neurologic puzzle.

2. As to eye diseases causing ear conditions I know only of one; the sympathetic ophthalmia, which is said to be the cause of deafness.

The first who observed this fact, Snellen, a. o., thought the deafness to be caused by a meningitis spreading from the optic nerve sheaths, because they believed in Deutschman's theory of the migration of the sympathetic ophthalmia along the optic nerve sheaths.

Nowadays this theory has become more or less obsolete and other theories are more favored.

Peters, who believes that deafness is not a very rare occurrence in sympathetic ophthalmia, explains the deafness by one of the modern theories and he chooses Elschnig's theory, which holds that the sympathetic ophthalmia is an anaphylactic process in sequence of sensibilization from pigment caused by the inflammation in the first eye. Now it is possible that just as the pigment in the second eye, also the pigment in the labyrinth becomes sensibilized and causes the ear disease. The individual difference in the amount of labyrinthine pigment can explain why deafness occurs in one and not in another.

I for one cannot say that I have often found deafness in sympathetic ophthalmia, but this theory shows again how close relation is thought to be present between eye and ear pigment, and I am thoroughly convinced that this relation is a very important one. We often encounter deafness in albinotic animals, for instance, white cats. As a rule these cats have a blue iris, which means that they are not absolutely albinotic, whereas total albinotic cats are not deaf. The same is true of white mice; totally albinotic white mice are not deaf, while the partial albinotic dancing mouse is deaf.

Concerning the relations between eye pigmentation and deafness there are several possibilities.

1. The pigment aberration causes the deafness.
2. The ear disease causes the pigment aberration.
3. Both are independent of each other and may come from the same origin.

The first is perhaps the case in sympathetic ophthalmia. As to the second possibility, Peters suggests, that the eye pigment may be sensitized from the labyrinth to a spontan-

eous bilateral iridocyclitis; this supposition lacks evidence up to the present.

Von Stein thought that the relation between eye pigment and labyrinth is so close a one that whenever he destroyed one labyrinth in a guinea pig he observed pigment changes in both eyes. Von Steyn could cause pigmentation as well as depigmentation of the eye by destruction of the cochlea, and supposed that there was in the cochlea a trophical center for the eye.

I cannot believe the relation between ear and eye pigment to be so close and simple, because Dr. De Kleyn and I did not observe a change in eye pigmentation in a great number of guinea pigs, rabbits and cats in which one labyrinth was destroyed for other reasons, either immediately nor some weeks or months after the operation. We had the same negative results in observing human beings, where the labyrinth was destroyed by diseases, so that we came to the conclusion, that pigmentation changes in the retina are often found in congenital but seldom in the acquired labyrinth deafness, and that it has not yet been proved that pigment changes are dependent upon the ear disease.

3. We have come to the third part of our subject, the influence of the ear on the eye. I will not discuss choked disc due to acoustic tumor or otogenic encephalitis or meningitis, but I will refer to some other diseases: In the first place thrombosis of the sinus cavernosus. Every ophthalmologist should always bear in mind that this usually fatal disease may have its origin in an otitis so that as soon as we can make the diagnosis or even suspect it, we ought to consult with an otologist, because, as I know from experience, operation upon the ear may sometimes prevent fatal issue.

Another eye symptom caused by an ear disease which occurs more frequently is the paralysis of the abducens nerve in otitis media. Some time ago I saw a most striking instance. A village physician sent a boy with paralysis of the right sixth nerve and stated that he had observed purulent secretion out of the right ear. When the boy came to Leiden the purulent secretion had stopped entirely and the otologists could not find the least symptom of otitis media, not even a scar or perforation in the tympanic membrane. The

boy was taken in my ward. Two days later the purulent secretion reappeared and the paresis improved, and a very small perforation was observed in the tympanic membrane. The secretion stopped again, the paresis became worse; this repeated sometimes.

What is the relation between the paralysis and the otitis? Gradenigo thinks that there is a meningitis at the top of the os petrosum; others consider it a pressure due to inflammatory edema and venous stasis in the narrow slit of Dorello, where the nervus abducens passes with the sinus petrosus superficialis. I for one believe that toxins play a part also by being distributed by spreading in the neighborhood, so that as soon as the free exit of the pus was blocked, the paresis was increased by toxic edema. It is like the affection of the optic nerve in disease of the accessory sinuses and like eye affections of dental origin.

According to my conviction, it is somewhat absurd to suppose that the toxins are absorbed by the fluids and run around the body to come exactly to the spot of origin next to the heart to exercise their pernicious influence.

It is much more probable and quite in accordance with the fact that the least obstruction causes immediate exacerbation to consider that the toxic influence comes directly from the toxins spreading in the neighborhood.

The combination of ear disease and abducens palsy may also be found in cases of traumatism. I present the roentgenogram of a man who, following a trauma of the head, became deaf and had an abducens paralysis. A fracture in the os petrosum passing through the cochlea is to be observed.

If we will remember that we have an otogenic pupillar and a palpebral reflex, we will discover in daily practice manifold relations between the eye and ear worthy of our cordial interest and valuable to our patients and ourselves.

Much more important than those direct clinical relations of the ear and eye are those between eye and vestibular organ, many of which have already found a place in the routine of our daily practice. They are not only of clinical interest, but also of high scientific significance, both from a physiologic as well as pathologic point of view.

THE VESTIBULAR ORGAN AND THE EYE.

The inner ear is composed of two different parts, which, though both are innervated by branches of the acoustic nerve, do not concern each other; the real auditory organ, the cochlea innervated by the *nervus cochlearis* and the vestibular organ innervated by the *nervus vestibularis*. Formerly the vestibular organ was thought to be a part of the auditory mechanism, and many names still recall this error to us. Since the classic experiments of Flourens, however, we know, that the vestibulae is a part of the organ of equilibrium and it is only the very close and intimate anatomic relation which gave this precious treasure to the otologists, although it has become the center of study and tremendous mutual interest for physiologists, neurologists and ophthalmologists as well as of otologists.

The vestibular organ consists of the three semicircular canals and the two spaces which we call the utricle and the saccule. In the ampullae of the semicircular canals we find the cristae acusticae, that are the nervous endings of the *nervus vestibularis* in nerve cells with motile hairs, moving with the motions of the lymph in which they are floating. Every movement of this fluid acts as an excitation to the nerve cells, so that those canals are the most excellent apparatus to perceive every motion of the head.

In the saccule and utricle we find the maculae acusticae, nerve cells on which rests a membrane with calciferous impregnation, the so-called otolith membrane, which in some animals, as fishes, is so developed that we can speak of a real stone, the otolith.

Otoliths may exercise influence on the maculae by pressing on the nerve cells or by pulling at them, consequently the stimulation which they cause will be altered by every change in position of the otolith organ, and thus the otoliths are an excellent organ to perceive every different position of the head in space. Numerous theories are brought forward and experiments performed to explain the function of the vestibular organ and I need only mention the names of Flourens, Mach, Breuer, Ewald, Krcidl, Kubo and Barany. You know how Barany applied our knowledge of the function of the vestibular organ for use in daily practice.

I think that some of the very best work done in this connection in the last ten years was in the pharmacologic laboratory in Utrecht by Magnus and De Kleyn and their co-workers. This is so important, that Barany himself is always in correspondence with these Utrecht men to hear whether his hypotheses are confirmed by the interesting experiments undertaken at that place.

The experiments were made on animals, frogs, cats, dogs, guinea pigs, but especially on rabbits, with the purpose of getting an exact knowledge of the function of the vestibular organ in those animals and thus to approach the problem in human beings, where the conditions are so quite otherwise.

The first thing which was necessary, was to be exactly acquainted with the anatomy of the vestibular organ of the rabbit.

In the anatomic institute in Utrecht de Burlet made serial slides of rabbit skulls, reconstructions in wax of skulls and membranous labyrinth; on the slides was made a mathematical drawing reconstruction, which was controlled by Prof. Ornstein with an analyticogeometrical formal system. In this way they succeeded in getting exact knowledge as to the anatomy of the labyrinth of the rabbit.

According to the result of this examination a magnified model of the labyrinth was constructed. It was too large to bring it with me but I can show you here a model of the otolith apparatus only, which represents their real position in the rabbit skull. The maculae acusticae are represented by these colored plates, the otoliths by these leaden ones. You see that in giving the rabbit skull different positions in space we can see what the position of the otolith is under any position of the skull. We know that the labyrinth may cause clonic and tonic reflexes on the eye muscles so that nystagmus is produced by the clonic reflex, whereas the position of the eye in the orbit is determined by the tonic. Now Magnus and De Kleyn found that the labyrinth produces also tonic reflexes on the body muscles and they tried to determine which reflexes were produced by the different parts of the vestibular organ.

They found that the vestibular organ causes maximal tonic contraction of the extensors of the limbs when the head of

the rabbit was placed topsy turvy, minimal when the head had its normal position with the mouth fissure horizontal.

This reflex was ascribed with high probability to the utriculus otolith so that we see the otolith causes the maximal extension of the limbs when it is dragging at the macula, no extension when it is pressing on the macula. The same otolith causes also extension of the neck muscles with, of course, the same maximum and minimum positions, but there is an important difference between both reflexes.

The utriculus otoliths are in contact with the limbs of both sides, so that when one labyrinth is destroyed the reflex rests the same in quality.

Concerning the neck muscles, on the contrary, the otolith is only in relation to the muscles on one side, so that when one labyrinth is destroyed the reflex causes bending of the neck.

A third labyrinth reflex results in the animal trying to bring back his head to the symmetrical medial position as soon as it has left this position. Magnus calls these reflexes *stell reflexes*.

They are probably caused by the main part of the sacculus otolith. Every sacculus otolith awakens a reflex, which tends to press the head in a direction opposite to the otolith. So long as the head is in the symmetrical medial position the influence of both otoliths counterbalance each other, but as soon as the head is bent to one side the otolith of that side is dragging more and thus stimulating more; the other is dragging less and thus stimulating less, so that the lowest otolith has the most influence and presses the head back again into the symmetrical position, which he cannot pass, for in that case the influence of the other is gaining in strength while the first loses its influence. If one labyrinth is destroyed the influence of the remaining sacculus otolith is no more counterbalanced, and it will press the head to the other side, until the head is in a horizontal position with the side of the remaining labyrinth above. Then the sacculus otolith presses on its macula, drags no more, so that this is the rest position, as was already determined by Flourens' experiments.

These reflexes have more than theoretical importance, for we can find them under circumstances in human beings, and they may be used for diagnosis. These reflexes are very dif-

difficult to discover in animals, and almost impossible in human beings, for the reason that they are obscured by voluntary movements and by other consequences of brain action.

They are found in animals the best when they are decerebrated. In human beings we find them when the brain does not yet act properly, as in some children under three and one-half months old, or when the brain is partly diseased. Magnus and De Kleyn have studied the course of the reflex, and thus we may conclude when the reflexes are present which part of the brain is still acting. These reflexes were found in patients with idiocy, meningitis, hydrocephalus, apoplexy and diabetic coma.

As a rule it is an ominous sign in brain disease when these reflexes can be observed.

We have seen that the vestibular organ can cause tonic and clonic contractions of the eye muscles. The first determines the position of the eye in the orbit.

Dr. De Kleyn and I tried to find out what are the different positions of the eye in the orbit when the head changes its position in space.

For this purpose we marked the cornea of a rabbit with a cross figure; suspended the rabbit in an operation board, the head fixed firmly in a Czermak clamp. At this clamp a wire figure was fixed, so that when we photographed the cornea we could see at the cross figure and the wire figure if and how much the cornea had moved. The photographic apparatus was fixed on the same board.

This operation board was fixed in a wooden frame in such a way that it could rotate on an axis and the frame again in another frame, so that it could rotate along an axis perpendicular to the first. In this way we could give the head of the animal every position in the space we wished.

When we turned the whole apparatus around a vertical axis the position of the eyes was not changed at all.

We made three rotations:

Rotation 1. Animal in vertical position, mouth fissure horizontal. Rotation of the animal on its bitemporal axis. Direction of rotation head down, tail up.

Rotation 2. Vertical position, mouth fissure horizontal. Rotation of the animal on its occipitocaudal axis.

Rotation 3. Animal in lateral position, mouth fissure vertical. Direction of rotation head down, tail up.

We made 25 photographs for every rotation of 360 degrees. The animal was moved every time 15 degrees, and we waited till nystagmus caused by the movement was over. At the twenty-fifth the animal had returned to its original position, so that the twenty-fifth photograph acted as control in determining if any change of the position due to other facts had taken place. It is clear that many of the positions in the three series are the same and so they controlled one the other.

In this way we learned that by changing the position of the head in space the eyes deviate typically in vertical directions and rotate along the sagittal axis. We could not find out typical horizontal deviation.

The vertical deviation has its maximum when the animal is rotated on his side; the eye of that side is as high as possible, the eye of the upper side as low as possible. De Kleyn and Magnus explained this by actions of the main part of the sacculus otolith. Every sacculus otolith is in connection with the rectus superior of its corresponding, the rectus inferior of its opposite eye. As long as the head is in a symmetrical position the influence of the muscles counterbalance each other, and so we see no vertical deviation, or practically none, when the animal is rotating around the bitemporal axis.

When, however, the animal leaves the medial position, the lower sacculus otolith drags more at its macula, the upper less, consequently the eye of the lower side is turned up, that of the upper side turned downward.

If one labyrinth is destroyed, the influence of the remaining sacculus otolith is not counterbalanced and the eye of the mutilated side is turned downwards, on the sound side upwards and the eyes return to the normal position only when the head is rotated on the mutilated side, so that the remaining sacculus otolith presses on its macula and so exercises no influence.

The rotation of the eye, the cyclotropia is maximal with the summit of the vertical to the nose when the nose is turned upwards; maximal with the summit to the ear when the nose is turned downwards. It was difficult to find out which oto-

lith produces this reflex, as neither the utriculus nor the main part of the sacculus otolith can do it. Now, Magnus and De Kleyn tried to explain it by action of the small bent part of the sacculus otolith. This part has not only a separate position nearer to the frontal plane, but also a separate innervation; whereas the main part of the macula sacculi is innervated by the nervus sacculus, this part is innervated by the nervus utriculus, so that it is no wonder when it has a separate action too. When the head is rotated with the nose vertically up in the air, the small part of the sacculus otolith drags at its macula and stimulates to the superior oblique muscles of both sides, so that the eyes rotate to the nose. When the head is rotated with the nose pointing downwards, these otoliths press on their maculæ, and this produces a stimulus for the inferior oblique of both sides. When one labyrinth is removed the reflex is the same in quality but not in quantity. The weak point of this explanation is that we must here accept for the first time pressing on the macula as a stimulus, but it is possible that this explanation is good, though I have some other objections.

This rotating reflex is called the compensatory rotation, because it seems to try to compensate the rotation of the head by the rotation of the eyes. That really the labyrinth is cause of these deviations is proved by the fact that when we destroy both labyrinths the compensatory and vertical movements have disappeared when we examine the influence of the head movement without bending the neck of the animal.

I could show that these reflexes are absent in the congenital deaf dancing mouse and also in two congenitally deaf and dumb girls. For the examination of the latter I used a Javal ophthalmometer with a rotatable head rest in which the head was fixed. We first determine the axis of the astigmatism of the cornea and then we rotate the head with the headrest, for instance, at 10° ; when there is no compensatory rotation the axis of astigmatism is 10° rotated. If there were compensatory rotation of 10° the axis would not have changed; if the axis was changed 3° there must have been 7° compensatory rotation, so we can determine exactly the compensatory rotation in astigmatic human beings, and in doing so we find that even the least rotation of the head produces compen-

satory rotation. This is of importance for the ophthalmologist, for it teaches us that we must always put our patients with their head straight in the ophthalmometer, otherwise we cannot find the right axis. This compensatory rotation is also probably the reason that astigmatic people often hold their head rotated to one or the other side when they do not get the right correction. They try by this movement to rotate their eyes in such a way that they get their eyes in the best position behind their glasses.

We as investigators ask ourselves what is the purpose of this compensatory rotation? When this rotation was observed years ago, they thought it was to hold the vertical meridian of the eye vertically in every position of the head for better orientation, but this could not be accepted because the compensatory rotation was always less than the rotation of the head.

In rabbits it is, as you saw, the same, but we know that there is another influence which gives compensatory rotation of the eyes, namely, a reflex produced by the neck muscles.

When the neck of a rabbit is bent, the eyes rotate. Now, De Kleyn examined both separately: the compensatory rotation caused by the neck, by fixing the head and bending the body of the animal toward the head, and the labyrinth rotative reflex in the way above described. When De Kleyn put both compensatory rotations together he learned, as you see, that a rabbit with the mouth fissure horizontal, head straight forward, can lift his head over 10° and bend it downwards over 90° without rotation of the eye, because the compensatory rotations compensate fully the rotations of the head.

At first De Kleyn was puzzled as to why this compensation allowed the head to bend more than 90° and only to lift 10° . But this was easily solved, for the normal position of a sitting rabbit is not with the mouth fissure horizontal, head straight forward, but with the head bent downwards 35° , so that out of this position the animal can lift the head over 45° and bend downwards more than 55° without the vertical meridian of the eye changing its direction, which is quite enough for orientation in looking for his food.

So we see that in rabbits the compensatory rotation corrects the influence of the rotation of the head fully, and it is probable that in mankind this is atavistic.

In discussing the highly interesting problem of the vestibular nystagmus, I must be brief and will only speak of one point.

You know there are many excitations which produce vestibular nystagmus; one of them is the irrigation of the ear with cold water, the so-called cold water nystagmus.

Bartels holds that this is caused by elimination of the labyrinth by the cold, so that the same nystagmus is produced if one labyrinth has been destroyed. Barany, on the other hand, thinks it is caused by a stream of endolymph in the semicircular canals brought about by local cooling of the labyrinth wall. This cools the lymph at that spot, consequently this fluid gets heavier and it flows off towards the lowest part of the semicircular canal. The lymph stream stimulates the sensory epithelium of the ampulla and causes in this way the nystagmus.

At first Magnus and De Kleyn tried to determine if such a great influence as paralyzing a nerve in the ear can result from cooling of the ear. Now De Kleyn had discovered that in cats the sympathetic nerve of the eye runs through the middle ear, and they observed that irrigating the ear with cold water caused a palsy of this nerve in cats, narrow pupil, narrow eye slit, etc., consequently Bartels' explanation is possible, but De Kleyn and Storm Van Leeuwen could prove it is not right, because the nystagmus does not act in the same way as the nystagmus after labyrinth extirpation. Later these two experimenters proved that Barany's theory was correct in the following way:

De Kleyn and Storm Van Leeuwen thought if Bartels is right the nystagmus will be the same in whatever direction the head is held. If, on the contrary, Barany is right, there will be an ampullapetal lymph stream when the ampulla is lower down, an ampullafugal stream when the ampulla is higher up than the cooled spot; no stream when the whole canal is horizontal—that means that the nystagmus must change abruptly in direction when the cooled spot crosses the position in which it is on the level with the ampulla.

To examine this, De Kleyn and Storm Van Leeuwen used our rotating operation board and irrigated one ear of a rabbit

during the rotation with cold water; they noted the direction of the nystagmus down after every 10° rotation so that the rotation of 360° gave 37 notations. You see here the result, which is corrected from the influence of the change of the direction of the internal rectus muscle by the rotation.

In no one of our three rotations does the horizontal canal really become horizontal, but we can reckon where the nystagmus must change its direction, and you see the result is very near to what was reckoned out before.

In this way it was proved that Barany's explanation is right. The cooling down of the horizontal semicircular canal plays the principal part in the genesis of cold water nystagmus.

If we review the different functions of the vestibular organ we find: Tonic reflexes on the body and neck muscles; Stell reflexes; tonic and clonic contraction of the eye muscles.

We always take for granted that the tonic reflexes are caused by the otolith apparatus, the clonic by the semicircular canals, but until now this has never been proved experimentally.

Whenever experimenters tried to take away the otoliths, the whole vestibular organ was spoiled, and the same was the case when they tried to perforate and drain the semicircular canals only. Now De Kleyn remembered that Wittmaak had published experiments in which he put guinea pigs in a centrifuge and revolved them about 2,000 rotations in a minute; then the centrifugal power was so great that the otolith membranes were torn and thrown away from the maculæ.

Magnus and De Kleyn made use of this. They examined a great number of guinea pigs on every vestibular reflex, then narcotized them, put them in the centrifuge and revolved during two minutes at 1,000 rotations a minute. As soon as the guinea pigs awakened from the narcosis they were again examined on the vestibular reflexes, and this examination was repeated during a couple of days till nothing changed any more. Then the animals were given to the anatomist, who examined the labyrinth in serial slides.

Magnus and De Kleyn had made the clinical diagnosis of what had happened in the labyrinth, which otoliths were torn and thrown away. The anatomist made the anatomic diagnosis. They knew nothing of each other's diagnosis till they

made comparisons, and in nearly every case the clinical diagnosis proved to be right.

I can show you here microphotographs of normal otolith apparatus of the rabbit, of otolith membranes detached from torn otolith membranes partly detached and partly in place, of vestibular organs with large hemorrhages after the centrifuging, and after some experience the clinical diagnosis could nearly always be confirmed.

In this way the function of the otoliths and of the semicircular canals was for the first time proved, and it was determined that otoliths are the organs for the reflexes of position, the semicircular canals for the reflexes of movement, but there was only one surprising fact.

The reflexes of progressive movements, as lift movements, etc., proved to be caused by the semicircular canals, whereas Breuer had always said this to be absolutely impossible, and everyone had believed him.

Magnus and De Kleyn have already explained this fact, but it would take me too long to discuss it.

We cannot say with absolute certainty if the explanation of the action of every particular otolith which Magnus and De Kleyn maintain is correct, but we know these explanations to be probable and the facts on which they are based to be true. Others try to explain the otolith action in another way—e. g., Quix, who explains it by considering the pressing of the otolith on the macula as the one acting stimulus.

Mr. Chairman, Ladies and Gentlemen—In the first part of my address I recalled to your memory the manifold relations between eye and ear which we may encounter in daily practice. In the second I dealt with one of the most difficult problems of medical science, and therefore I do not flatter myself that every one of you will have understood everything I tried to express, but I am certain I have given you something to think about. You see that we are not at the end of all the problems, how after every solved problem new questions arise which await their solution, but I hope you are convinced with me that we are on the right track and will, in going on in this way, come to know as much of the functions of the vestibular organ, as much of the relations between labyrinth and eye, as is given to human beings to know.

XLVIII.

METASTATIC LARYNGEAL ARTHRITIS.

By S. O. FIELDS, M. D.,

NORFOLK.

From the paucity of the literature on the subject of joint affections of the larynx one is almost forced to conclude that the condition is either an extremely rare one or else that observers do not make the diagnosis often owing to certain inherent difficulties in and obstacles to accurate observation. Taking into consideration the fact that there exists no insurmountable pathologic or anatomic reason why the laryngeal joints should not be subject to the metastatic conditions affecting the other joints of the body, the conclusion is obvious that the rarity of the affection is merely apparent rather than real, and that the difficulty lies in properly interpreting the often indefinite and elusive symptoms and in correctly translating the not infrequently vague physical manifestations.

The etiologic factors of the condition are manifold at first glance, but a simplification is possible if these causative factors are regarded from three fundamental viewpoints. First, we meet with the affection in such general infections as typhoid, influenza, syphilis, tuberculosis. Secondly, it occurs as a concomitant manifestation of involvement of contiguous tissue, as in the cases in which a perichondritic or phlegmonous process makes itself felt in the joints of the larynx. Thirdly and most important of all, the condition is encountered as a local manifestation of a distant infectious process. This last view, or view of metastatic focal infection, occupies the most important position in the etiologic scale and harmonizes entirely with what we know of similar processes taking place in the other articular structures of the body. It is as logical that the joint structures of the larynx should be involved in an individual with infected tonsil, nasal sinus trouble, abscessed teeth, or a posterior urethritis, etc., as it is for the knee or elbow joint to be likewise attacked under similar circumstances. That in these metastatic cases there

is some additional factor determining the localization of the bacterial activity to the laryngeal joints aside from a selective affinity of the causative organisms we feel reasonably certain; for in three of our four cases herein reported it was only after the occurrence of intercurrent factors producing probably a more or less marked hyperemic state in the larynx that the joint manifestations were evident. So it is likely then that in addition to having the selective bacterial agents we must also have the proper preparation of the soil.

When we come to the pathologic features characteristic of laryngeal articular involvement we are forced to confess, if we are frank, that our knowledge is woefully deficient. Not enough postmortem work has been done in the condition to render us any appreciable amount of help. We know that the cricoarytenoid joint is affected in the vast majority of the cases which present themselves. Sometimes the affection of the laryngeal joint is found alone and sometimes it is seen as an accompaniment of a general polyarthritic condition. Reasoning from analogy and from our knowledge of similar processes in the other joint structures of the body, there is little doubt that we have the characteristic serous exudation into the joint cavity, the typical round cell infiltration, the hyperemia, the swelling, often the infiltration of the periarticular tissues, and if the process is a chronic one the deposition of cicatricial or connective tissue, within or without the joint capsule, often going on to partial or total ankylosis.

The symptoms of the condition are in not a few instances so vague and indefinite that their correct interpretation presents quite a problem. In character they range from manifestations which are only suggestive to those which are well marked and clear cut. On the one hand, there are met cases presenting solely a history of paresthesia of the laryngeal region and a variety of indefinite complaints, and on the other are encountered cases which present every type of pronounced laryngeal irregularity—e. g., pain, hoarseness, dyspnea, etc. The patients in the less severe cases complain simply of a sensation of fullness or tension or itchiness in the throat, situated in the lateral region of the larynx and aggravated especially by swallowing or speaking efforts or on touching the spot with a probe. In addition the patient may com-

plain of a sensation of using the voice under a strain. In nearly every case, whether mild or severe, there can be obtained the history of some event or occurrence about three or four days prior to the laryngeal joint manifestation of sufficient degree to provoke a hyperemia or irritation of the laryngeal mucous membrane. The pain encountered in the more severe cases is often quite intense and manifests a tendency to radiate upward into the ear or downward along the sternomastoid on the affected side, but in our experience this pain does not approach in severity the type encountered in phlegmonous laryngitis or in perichondritis. Neither is the hoarseness so pronounced or the dyspnea so marked as in the latter affections. A typical feature of the affection which was present to a greater or less degree in our cases was the so-called Grünwald's crepitation, this sign being elicited by intermittent pressure on the upper and back part of the thyroid cartilage and being characterized by a peculiar grating or "moist rale" sensation which can be both felt and heard, this sound owing its origin to the rubbing together of the inflamed articular lining.

On examining the larynx we find in the majority of cases a slight amount of redness over the arytenoid cartilage and also a moderate amount of swelling. Both the reddening and the swelling vary according to the severity of the affection, but in most instances they are not very marked, and the redness in most cases presents in addition a rather streaky appearance in striking contrast to the diffuse reddening noted in other conditions affecting the region of the arytenoid cartilages. The vocal cord shows impaired motility and not infrequently presents a jerkiness of motion which is evidently due to the increased friction between the roughened synovial surfaces. Absolute fixation of the cord and the presence of scars and adhesions are evidences of a severe process which has lasted a long time and are seldom seen as a result of metastatic laryngeal joint trouble.

Though it is fairly difficult in some cases to differentiate the condition from perichondritis, paralysis of the cord, etc., still careful analysis of the history and keen observation of the course of the process render a correct diagnosis by no means a matter of impossibility. It is highly important to

remember that these joint affections, like similar affections in the other joints of the body, have in the majority of instances, a primary focus of infection to which they owe their origin. The acuter cases are prone to undergo resolution subject to recurrences, unless through careful search we ascertain the original site of infection, but if the process enters upon a chronic adhesive stage or if it is of that type from the beginning we are likely to have as a result adhesion, stenosis and other disastrous sequelæ.

The following cases are likely to prove both interesting and instructive because they represent, with one exception, types of laryngeal joint affection due to metastatic infection:

Case 1.—J. L., male, age twenty-eight, single, negative family and past medical history, came from Margarettsville, N. C., to the Princess Anne Avenue Clinic, complaining of pain in the right side of the larynx on swallowing or speaking, and marked sensitiveness to pressure over the upper and outer part of the thyroid cartilage. He stated that six days previous to his first visit he had attempted to inhale steam from very hot water for a condition which his physician had called "catarrh in the head," and the vapor being almost scalding had pained him severely. He paid no attention to the matter, for it seemed to be all right in a few hours. About three days afterward his present throat trouble developed. His voice, while not strictly hoarse, exhibited a roughness or huskiness which caused him to clear his throat constantly in the effort to raise what seemed to him obstructing mucus, thereby aggravating the existing pain. Laryngoscopically there were found slight swelling, delimited to the region of the right arytenoid, and a little streaky reddening over the same structure. The vocal cord exhibited characteristic jerky movements. There was tenderness on pressure, and Grünwald's sign was easily elicited. Urine, pus threads. Wassermann, negative. General examination negative save for a moderate degree of swelling and pain in the right knee which came on about ten days ago, three weeks subsequent to the onset of a urethral discharge diagnosed as clap. Staining and microscopic examination of a centrifuged specimen revealed the gonococcus. Following the institution of treatment for his urethritis, combined with the use of vaccines for his gonor-

rheal knee, the throat condition rapidly improved, and in two weeks he reported himself normal, both as to knee joint and throat. An examination made at this time showed normal cord motility, an absence of pressure tenderness and the disappearance of Grünwald's sign.

Case 2.—E. S., female, high school pupil, age 17, subject to recurrent attacks of tonsillar inflammation, tonsils being of the submerged buried type, crypts containing pus. Sometimes during her tonsillar attacks she experienced pain in her knees, elbows and wrists. Past medical and family history irrelevant. Wassermann, negative. Urine, normal. General physical examination, negative. Five days before coming under observation, while imitating another schoolmate, she had attempted to smoke a cigarette. She states that her efforts were only indifferently successful, for nearly all the smoke "went down into her windpipe" every time she attempted to blow it through her nostrils, and she sputtered and coughed and choked for nearly fifteen minutes before abandoning her maiden effort to be a devotee of modern fashion. She dismissed the matter from her mind and thought no more of it until two days later, when she noticed that the region of her larynx was painful and tender to pressure. The pain of which she complained ran down along her sternomastoid on the left side. While her speech was not distinctly hoarse she showed a coarsening of a voice which was normally most delightful to hear. Her tonsils at this time showed evidences of a recurrent inflammation, and in addition she complained of slight fugitive pains in both elbows and knees. Laryngoscopically the typical slight reddening and the moderate arytenoid swelling were visible. The movements of the right cord were normal, but those of the left were much inhibited. Grünwald's sign was obtained. Despite the subacute process in the tonsils they were removed, with immediate improvement in the patient's laryngeal condition and the disappearance of the pains in her other joints. Since that time she has had no more trouble.

Case 3.—C. H., female, married, age 48, had experienced annoying pain and sensitiveness in her right upper canine tooth ever since a dentist had put in a filling a year before. She also suffered severely with "rheumatism," so that at

times she was well nigh incapacitated from the pain and stiffness in her joints. She came into the throat department of the clinic complaining of a peculiar itching and drawing sensation in the right side of her larynx, which at times amounted almost to actual pain. Though her husband stated that he could detect a slight change in the timbre of her voice, she herself asserted that it was not pronounced, and that her chief concern was the unpleasant feeling in her throat. Past medical and family history negative. Wassermann negative. Urine normal. General physical examination negative. Deep pressure over the thyroid cartilage showed slight and questionable tenderness on the right side. Grünwald's sign present. Characteristic reddening and swelling over the right arytenoid. Almost no impairment of motility of the vocal cord, although the right cord showed a moderate amount of jerkiness in its movements. Finding nothing to account for her general or laryngeal joint manifestations save the sensitive tooth, an X-ray was made, and this revealed a fairly large apical abscess. Removal of the tooth was followed by progressive improvement in every respect, and at her last visit, three weeks later, the patient stated that she felt better than ever before in her life. Laryngoscopic examination at this time revealed a perfectly normal condition of the laryngeal structures.

Case 4.—P. T., widow, age 52, came under observation July 8, 1921. History of several miscarriages. She stated that for several years she had suffered from vague fleeting pains and aches in all her joints and muscles. About five days previously, so she stated, she swallowed a large drink of tea "the wrong way," and as a result she had a severe coughing and choking spell for nearly half an hour. Two days afterwards she noticed that the region of her larynx was moderately painful on swallowing and speaking, while there was considerable soreness on both sides when she made pressure on the painful area. Her voice was moderately husky. Grünwald's sign easily elicited. Laryngoscopically the moderate swelling and arytenoid reddening were noted, bilateral in this case. Slightly impaired motility of both cords. General physical examination negative. Urine normal. Wassermann plus four. Despite the most thorough search no focus of infection was discoverable. Intensive antiluetic treatment was then begun, on the

basis of the positive Wassermann and the history of many miscarriages. In ten days her laryngeal symptoms had vanished, followed soon by the disappearance of the vague joint and muscle discomfort. When last seen, about six months ago, her larynx presented a normal condition and she asserted that she felt like a new woman.

An analysis of these four cases reveals in every instance—even in the luetic case—that the actual onset of laryngeal articular trouble was preceded by some occurrence which probably sufficed to render the laryngeal structures a locus minoris resistentiae. It also serves to emphasize the fact that focal infections are to be taken more into consideration in these cases, thus broadening the scope of a field already of no inconsiderable magnitude. Furthermore, it shows the necessity of taking up the subject of laryngeal joint affections again and revamping many of the pet theories which we have held all along concerning the matter. If, in this imperfect and preliminary study, we have succeeded in focusing a little attention on the subject we shall feel that our efforts have not been in vain.

1008 CHURCH STREET.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Wednesday, November 1, 1922.

THE PRESIDENT, DR. CHARLES H. LONG, IN THE CHAIR.

PROF. ROBERT BÁRÁNY, Upsala University, Sweden, delivered an address entitled:

"Oto-Neurology and the Necessity of Co-operation Between Ear and Eye Specialists, Neurologists and Brain Surgeons."

Professor Bárány took up, first, the historical development of various tests in connection with otologic and ophthalmic work. He stated that the anatomy of the vestibular apparatus had been known for several hundred years. Many sane observations had been made, but there were many speculations. For instance, during an attack of vertigo involuntary eye movements occurred, but no one in those days took the trouble to investigate why these eye movements occurred. Purkinje had the opportunity to observe the eye movements in vertigo in cases of insane people, who were placed in cages and turned around until they became nauseated, and eventually they quieted down. While this treatment was said to be effective, it did not seem humane. Purkinje studied the eye movements of these patients and found that under normal conditions eye movements were obtained by holding the head in different positions, and different results were secured, and Purkinje came to the conclusion that it was the head which was directly influenced by the drum. But he drew a wrong conclusion; it was the brain which was irritated by the drum. There must be a sense organ before the brain could get a sensation, and Purkinje thought the brain got the sensation directly. In the same year that Purkinje was doing his work Fleuron made some experiments on pigeons. He dissected the different canals in pigeons and found that when he dissected the horizontal canal, which lies in a horizontal plane, the pigeon turned in a horizontal plane; that if he dissected the vertical canal, the pigeon

would make curious movements which he could not understand. He did not think these birds were dizzy, and that it was vertigo which produced these movements. Purkinje was familiar with dizziness in man but not the dizziness of animals. He came near making a discovery, and if he had turned one animal he would have seen the difference between the dizziness of man and the dizziness of animals. Other men repeated the experiments of Purkinje and Fleuron without discovering anything especially new. In the year 1861 Meniere's disease was brought to the attention of the profession. At that time Meniere was sixty years of age, and his discovery was considered unusual but it was accepted. He had practiced as an ear specialist for many years, and frequently saw cases in which dizziness occurred. At this time, if patients presented themselves with attacks of dizziness he immediately made a diagnosis of cerebellar tumor. He had made several autopsies in such cases and had found cerebellar tumor in some of the cases. Meniere studied his cases carefully and found some patients who had had dizziness for thirty years but who never developed cerebellar tumor, so that he came to the conclusion that his diagnosis of cerebellar tumor was incorrect when based on dizziness alone. He began to look for some other explanation and found these cases always had dizziness, tinnitus and earache, although the ear drums were normal. The tuning fork was unknown at that time, but Meniere concluded from these other symptoms that these patients must have some lesion of the inner ear. The cochlea was for hearing, but it was not known what importance the semicircular canals had. Meniere received inspiration from the experiments of Fleuron and believed dizziness was the cause of the movements of the eye in animals without ever having turned an animal or examined it himself. He came to the conclusion that the dizziness of patients was due to some disease in the semicircular canals. It was subsequently pointed out that he was right in reaching that conclusion. He published several articles on what is known as Meniere's disease prior to his death. It was thought that Meniere's death did much to promulgate his theory, because Dr. Bárány believed that if he had not died his colleagues probably would have fought him, but instead they made a national hero of him.

In 1870 a German physiologist by the name of Glotz repeated the experiments of Fleuron and advanced the theory that if the semicircular canals were diseased the animals lost their equilibrium; therefore, it was probable that the function of the semicircular canals was to maintain equilibrium. Other men who made observations and experiments were Breuer and Mock of Vienna and Brown in America. Brown in turning animals saw that he produced the same movements as by dissecting the semicircular canals. He maintained that irritation of these canals was the cause of dizziness, because it was very clear the animals became dizzy. On the other hand, if he destroyed the canals on both sides the animals were again made dizzy. He advanced the theory that the movements of the endolymph, produced by turning, were the cause of irritation or of stimulation of the canals. Mock came to the same conclusion by other experiments which he made, but in a logical and philosophic way concluded that all the other causes could be excluded, and only the semicircular canals remained to produce the sensation of turning. Brown examined normal individuals with turning sensation in different positions of the head and said there could be no question that it must be the semicircular canals which produced sensation because the brain could not directly take its position. These men were not ear specialists, consequently ear specialists for a long time did not appreciate this theory on the ground that they had not found pathologic cases. Some examinations were made of deaf-mutes after the turning method of Breuer, but except for these findings there was not much learned. Quite a number of men had made valuable observations; among them Jansen of Berlin, who saw a large number of cases of fistula or lesions of the labyrinth, and was one of the first to advocate operating on the labyrinth when it was suppurating.

When the speaker began to examine these patients he had received three years' training in the clinic of Politzer of Vienna and had studied the literature on the vestibular apparatus in which he became very much interested. At the clinic he syringed those cases that had fistula of the attic of the tympanum and in so doing observed the patients became dizzy. From what he had read of the work of Breuer, it was clear to him in looking at the eyes that nystagmus was present. When

he syringed the right ear he noticed nystagmus to the left. This observation was noted on a sheet of paper which he put in his pocket. The next day he had another case of nystagmus which was treated in a similar way. He recorded this observation on a sheet of paper and put it in his pocket. In a short time he found he had something like twenty such cases that he had recorded and was astonished to find that he had always observed the same thing. He found that if he syringed once on the right side, and then the left side, he became confused, and it was clear to him that there was a law behind this, but he had no idea what that law might be. One day one of his patients came to the office and when he syringed his ear again the patient exclaimed, "Doctor, the water is too cold; when I syringe the ear myself at home I never get dizzy." The speaker then said to the nurse that the patient had complained of the water being too cold. The nurse in reply said it was not true; the water was not cold, as she had tested it with her finger.

The speaker pointed out the necessity of having the water warm. Subsequently the nurse made the water very warm and, after syringing the ear again, the patient exclaimed, "Doctor, I am again dizzy; the water is too hot." As Professor Bárány looked at the patient's eyes he immediately saw that the water had produced the opposite nystagmus. It was the temperature of the water which had produced this opposite nystagmus. This observation was a step to other conclusions. If it was the temperature of the water which produced this effect in suppurative otitis media, might not this phenomenon occur in other cases? He examined other cases after syringing with cold and hot water and found that all cases with normal drums showed the same phenomenon, only it took a little longer time to get it, as in a case where there was no drum. Up to this time he did not know how cold and hot water could influence the labyrinth to produce this nystagmus, but one day he saw an explanation for it. (Here Professor Bárány with the aid of blackboard diagrams showed how hot and cold water could influence the labyrinth sufficiently to produce nystagmus.) He found that if the labyrinth on one side was destroyed he did not get this reaction, so he sought for cases that had no reaction and he found such cases with no caloric re-

action, and when operated on it was found the labyrinth was destroyed, with the presence of cholesteatoma. The clinical value of the test became evident to him, and it was now possible to examine every vestibular apparatus. If one syringed the right ear with cold water, nystagmus was produced, the patient became dizzy, so that when he closed his eyes the sensation was of turning to the left side. He found subjective dizziness on turning sensations.

There were reflex reactions upon other muscles of the body besides the nystagmus. Such a nystagmus could be observed in man. If he got a falling reaction he could not stand erect with his feet together in the Romberg position; he would slowly fall to the right side. If his head was turned to the left side the falling was changed; he fell forward. If he turned his head to the right side he fell backward.

In thinking about the mechanism of the falling reaction, he employed a method which he found had been developed by Exner, a physiologist, who had written a book on the subject. Exner, he said, tried to construct mechanisms in the brain or in the spinal cord which would produce physiologic effects. These mechanisms were constructed upon the neuron theory. The speaker had found this method of Exner's valuable for constructing mechanisms in the brain and also for the vestibular apparatus. Possibly somewhere in the brain there was a cell which received its impression, and through this cell there was innervation down to the spinal cord, so that the patient fell to the right side. There was some stimulation of the semicircular canals during the turning of the head which produced this change. He found that if he held the head in the same position and turned the body and not the head, he got the same change, so that the cause of this change was not in the head, it must be in the connections between the head and trunk, and he came to the conclusion that it must be in the deep sensibility of the neck. After making other observations and tests he saw there was a double influence exerted on one center, and the question was, Where was this center situated? This center received impulses from the semicircular canals and impulses from the deep sensibility of the neck.

He then studied the literature on the histology of the brain to see if he could find where this center might be. He thought

it might be the principal nucleus of the vestibular nerve, but he could not find this center in the deep sensibility of the neck. He found, however, the deep sensibility of the neck extended into the cerebellum, and therefore he concluded that it extended into the vestibular cerebellum and not into the medulla oblongata. If the vestibular mechanism was destroyed he was not able to get this influence. Then he went to work to find out if there was disease in the cerebellum. He found such a case on which an autopsy was held, showing a tumor in the cerebellum. Several months later he was called upon to make a diagnosis in a case where neurologists could not find out on which side of the cerebellum the tumor was situated, and he was asked to find it out by his method of examining the right and left ears. After making tests he was not sure himself on which side the tumor was located, but finally decided it was on the right side. It was subsequently found that the tumor was on the left side.

He began to study the literature on the subject and found an interesting book by Bolk on the cerebellums of mammalia. In this book Bolk presumed that the cerebellum had something to do with innervation of the muscles and compared the cerebellums of different mammalia with each other. He found the giraffe had an enormous neck, well developed, while the neck was nearly absent in the mole, and he concluded that this part had to do with the innervation of the neck. After comparing the cerebellums of different animals he concluded that the hemispheres of the cerebellum must have something to do with the innervation of the extremities, and the middle part of the cerebellum had to do with the innervation of the trunk. In one case he found a tumor was situated in one hemisphere and not in the vermis, and therefore could not make the diagnosis before operation. By the pointing test he found innervation of the extremities in the same way as innervation of the musculature of the trunk and falling reaction.

The pointing test and falling reaction were not such simple reactions as nystagmus. Nystagmus was quite independent of the will of a person. Fixation could diminish the nystagmus and sometimes inhibit it, but if fixation was excluded one could see nystagmus exactly as it was without any influence upon the side of the person. The falling reaction was influ-

enced by voluntary movements. If we got such innervation in the right side, a person could fall to the left side, so that the reflex movements were not so easy of investigation as nystagmus.

Pathologic ear cases were more frequent than cerebellar lesions. Tumor cases were difficult because the tumor was only damaging the parts where it was situated, but the whole brain was being pressed upon in different directions, producing hydrocephalus and a number of symptoms, so that these cases were not so simple as the ear reactions in diseases of peripheral organs. Therefore it was necessary to have the cooperation and collaboration of many men to develop this work. There was still an enormous amount of work to do which would take many years, and specialists should cooperate in every way in carrying out further research work, which would eventually result in not only great benefit to the patients themselves but to mankind in general.

DISCUSSION.

DR. GEORGE W. HALL said that Professor Bárány had rightly stated the difficulty of exact localization in cases of brain tumor because of pressure in distant parts of the brain, and he would like to ask him for information what light he could give from his tests in cases of occlusion of the posterior inferior cerebellar artery, which causes certain vestibular manifestations?

PROFESSOR BÁRÁNY replied that he had examined two or three cases of occlusion of the posterior inferior cerebellar artery, and in one case there was marked nystagmus, which was to be explained by a lesion in the medulla, which occurred near the nucleus descendens of the vestibular nerve. He had seen severe disturbances of equilibrium in these cases, and the question arose whether it was the nucleus which was affected, which had connection down to the spinal cord, or whether it might be a lesion extending into the cerebellum and causing trouble there. He had also observed that certain pointing reactions were not present in such cases. If it was a lesion of the cerebellum or of the tract traveling down into the medulla oblongata, if certain pointing reactions were not present, then probably the lesion was in the cerebellum and hemisphere, and not in the medulla oblongata.

He had not been able to make autopsies on any of his cases, but he mentioned one case reported in the literature by a German in which a postmortem was made and the findings corroborated the symptoms manifested during life. In most of the cases that came to postmortem he said these tests had not been made; if they were made, they were incomplete, and it was not possible to reach a correct conclusion if one did not make a complete examination. In some of the cases he had seen reported in the English literature, in which spontaneous pointing was found and eye pointing reactions to the turning or syringing, there was only an examination of pointing in one plane and not in the other. Incomplete examinations did not help very much. In such cases there must be an exact examination, and then it would be interesting to compare these examinations with the autopsy findings. These cases were suitable for drawing deductions because the tumors were not pressing and making other symptoms.

DR. GEORGE E. SHAMBAUGH stated that there was one practical question which often confronted a physician, and that was the question as to whether in cases of vertigo the disturbance arose from the internal ear or from an intracranial disease. It must be quite clear to everyone present, from what Professor Bárány has just said, that vertigo and disturbance of equilibrium could be produced as readily from diseases of the posterior brain fossa as from alterations in the internal ear. Dr. Shambaugh believed that the otologist was able with very few exceptions to determine definitely whether the disturbance of equilibrium arose from internal ear disease or whether it was being caused by intracranial trouble.

This was always the first important question to determine in the cases of vertigo. He was not so confident that our interpretation of the reactions obtained by applying the Bárány tests, as they were now called, had reached such a point that we were able in many cases to determine anything more than that we had to deal with a disease in the posterior brain fossa. His experience had convinced him that these tests, when properly applied, and where they were properly understood, would often give a definite clew regarding the existence of some obscure intracranial condition much earlier than the neurologist could find any evidence of these conditions.

DR. H. I. DAVIS said it was not easy for the neurologist to answer certain questions, as was proven in a case seen by Professor Bárány and himself. The patient had had attacks of dizziness and disturbances of equilibrium and at one time mild epileptic seizures were suspected. On investigation the patient was satisfied that she had never lost consciousness during any of the attacks, and after repeated examination it was thought that the labyrinth might be responsible for them. When the patient was carefully examined by Professor Bárány the presence of moderate nystagmus was disclosed, and the question was settled in favor of the labyrinth.

DR. ARCHIBALD CHURCH was gratified to learn that even Professor Bárány still found difficulty in the interpretation of these complexes. The speaker had the impression that in one or two conditions a definite combination of symptoms developed. In these caloric tests, the turning test, the pointing test, and falling inclination were quite precise, as, for instance, in tumors of the pontine angle—the acoustic tumor in particular. He had a feeling that the neurologist picked out these cases as soon as the otologist, and he recalled several patients whom otologists had accused of having cerebellar and brain tumors because of some modifications in the responses induced by the Bárány tests. These patients had some bad moments under the impression gathered from the otologists that they were the possessors of brain tumors. However, this supposition was not verified by the clinical histories extending over a period of a year and a half, two and three years, respectively. The interpretation of these signs and symptoms was extremely difficult, and what at first promised to give an easy avenue to correct diagnosis of a localizing character, he was afraid in the great majority of cases only served either to confirm the neurologic observations or else to confuse them. The subject was one in the study of which all should cooperate. The fruits only would be gathered after much data had been carefully collected and thoroughly sifted.

DR. J. HOLINGER thought every one, after listening to the remarks of Professor Bárány, got the impression that it would take an enormous amount of joint work on the part of pathologist, neurologist, physiologist, ophthalmologist and anatomist in order to work out all the questions that were involved.

He believed we have no facilities to undertake such work. Many beautiful views had been opened up in various directions, but we have no men, no clinics or laboratories to undertake these investigations. The facilities for doing such research work are not at our command either in Chicago or in any other great medical center in the United States. It was high time for specialists to begin to work jointly in order to get an insight into the complicated workings of the brain, eye and ear for the best welfare of patients and of humanity in general.

DR. CLARENCE A. NEYMANN inquired of Professor Bárány about those cases of psychogenic pseudonystagmus with certain of the other reactions that recover or are improved after psychoanalysis. He recalled the case of a young girl whom Professor Bárány had seen in St. Louis. This girl showed certain reactions as to past pointing, etc. She was totally incapacitated for work until a definite psychic complex was unearthed. She still showed some reactions, but improved as regards dizzy spells and was able to work.

He had had several other cases in which there were attacks of tinnitus aurium, dizziness or vertigo that had done remarkably well under this sort of treatment, and he asked Professor Bárány if any psychic influence could change these tests.

PROFESSOR BARANY, in replying to Dr. Neymann and in closing the discussion, stated that he had never seen hysterical nystagmus. It was possible, however, that hysterical patients could produce eye phenomena, such as convergence, which was not at all infrequent. It was possible for a patient to produce voluntary nystagmus. He knew of men who could produce voluntary nystagmus and he could do it himself.

Another thing: hysteria was produced by disease of the vestibular apparatus very often, and then it was possible by treatment to improve the hysterical symptoms. For instance, he had seen a woman with moderate attacks of nystagmus, and by stimulating eye movements from the vestibular region she felt very dizzy. It was possible by some means to get rid of this sensibility to nystagmus.

As to the girl he saw in St. Louis, the patient was not cured by psychoanalysis. She had quite definite trouble when he saw her after treatment. Probably her hysterical psychosis had been improved, and one could by treating the hysterical

psychosis or hysterical symptoms get improvement, but a real hysterical nystagmus he had not seen. It was quite another thing with the past pointing which could be produced voluntarily. One could past point and this could be influenced by hypnosis and by other methods. He had a patient in Upsala who had dizzy spells with nystagmus and past pointing. One day he got the idea of putting a noise apparatus in her ear, which he did, and then the past pointing completely disappeared. In another case he used a tuning fork on the skull and the past pointing disappeared. The past pointing could also be made to disappear by psychoanalysis.

He did not believe that hysteria was a disease situated in the medulla; that hysteria was a subcortical disease and was not situated in the medulla oblongata, nor in the vestibular nerve. One could detect spontaneous nystagmus by tests, also spontaneous past pointing. There was a diminution of certain reactions in tumor cases where neurologists had not found anything. He saw such a case recently at the Mayo Clinic.

If one desired to make an accurate diagnosis in these cases he must be familiar with the various apparatuses: otherwise he would make mistakes.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, December 4, 1922.

THE PRESIDENT, DR. C. H. LONG, IN THE CHAIR.

Impaired Voice.

DR. ELMER L. KENYON presented a young woman of twenty-seven, a teacher, who had had a uniformly impaired voice for about twenty years. No physical cause in the larynx was evident excepting a bowed action of the vocal cords on phonation. The case would ordinarily be classified as a functional neurosis of the intrinsic muscles of the larynx. But since the intrinsic muscles could not function without cooperation of the extrinsic laryngeal musculature the old habit of studying only the action of the intrinsic musculature in such cases was evidently wrong. In fact, in this case the action of the extrinsic musculature was also evidently imperfect. Dr. Kenyon believed the trouble to be one of imperfection of vocal technic acquired in childhood and that the trouble was correctable by vocal training.

The patient had an acute illness when she was six or seven years old and before that had sung in a very high voice. Possibly during the acute illness she had had a laryngitis and had found it necessary to strain the entire musculature to produce an intelligible voice. As this continued for some time, she may have formed a bad habit of handling the voice and this habit may have persisted. Dr. Kenyon believed that in functional cases one should obtain accurate information as to the manner in which the extrinsic musculature varies in action from the normal and in this way such cases may possibly be placed upon a firm basis of understanding.

DISCUSSION.

DR. NORVAL H. PIERCE thought Dr. Kenyon opened up a very interesting field in his attempt to study the extrinsic muscles in the act of phonation. There was no question as to the fact that they had much to do, especially with the singing voice, but he thought it would be difficult to tell whether they were

acting properly or not. It was quite easy to tell the actions of the intrinsic muscles. It was not so easy to determine the action of the extrinsic.

DR. KENYON said that in the falsetto voice of puberty and in dysphonia spastica one could observe a definite disturbance in the action of the extrinsic musculature of the larynx. In certain other disorders of voice, like, for example, the one in question, the tendency to imperfect action was also clear, but not so positive. Since the intrinsic muscles could not function excepting with the cooperation of the extrinsic, the old habit of studying only the action of the intrinsic muscles for an explanation of their imperfection of action was evidently wrong. If one is to solve functional disturbances of the voice it must be by observation of the complete musculature on which vocal cord action depends, and not by observing merely one phase of such action.

One studies, mostly by palpation, the action relationship between thyroid cartilage and hyoid bone, and between these structures and the base of the tongue, the sternum and the spinal column. Definite sounds are given for purposes of such observation. Dr. Kenyon was attempting to accumulate facts in a variety of functional vocal disturbances. The X-ray might eventually be of service. In the patient in question he believed that vocal training would bring about recovery.

Ozena.

DR. ALFRED RUNDSTROM presented two cases of ozena, the first case one of genuine ozena, and the second one of hereditary luetic ozena.

Dr. Rundstrom saw the first case for the first time two years ago, and at that time the nasal cavities on both sides were filled with fetid crusts. The meati were wider than normal; the inferior turbinate bone was already atrophic, the atrophy more apparent on the skeleton than on the mucous membrane. He had removed the concha media on both sides and opened the ethmoid cells and the sphenoid sinuses on both sides. In all these sinuses there was a carionecrotic process. The left side at this time was perfectly healed. On the right side there was still some secretion, but the naked bones were now partly covered with mucous membrane, partly with granulations.

The second case, as already stated, was one of hereditary luetic ozena. In this case also the ethmoid cells and the sphenoid sinuses on both sides were the seat of a carionecrotic process. Dr. Rundstrom stated that these two forms of ozena resembled one another very much, although they were different as to etiology. This confusion happened so often that some authors, as was well known, claimed that genuine ozena always developed on a luetic basis. Resemblance consisted in the fetor and the carionecrotic process. The difference was that in the luetic ozena the carionecrotic process causes greater destruction and that these destructions may also be found in the nasal cavity, while in genuine ozena they are found only in the sinuses. While the carionecrotic process was present in both forms of the ozena it was caused in the first by pus infection and in the second by a luetic infection.

DR. MILLARD F. ARBUCKLE, St. Louis, addressed the society on

"Complications of the Naso-Sinuses."

ABSTRACT.

DR. ARBUCKLE said that on October 20, 1922, he presented to the St. Louis Pediatric Society a few cases of acute parenchymatous nephritis, sometimes called nephrosis, in which the results following treatment in suppurative diseases in the nasal accessory sinuses had been sufficiently noteworthy to justify reporting them.

They have had about twelve cases at the St. Louis Children's Hospital and in private practice. In these cases there was marked general anasarca, albumin in large quantities in the urine, casts and red and white blood cells also present in some. In all the cases, except one, suppurative disease in the nasal accessory sinuses was found.

In view of subsequent experience, he believed there was sufficient evidence in that case to justify the diagnosis of ethmoiditis and sphenoiditis, since the mucosa in the olfactory fissure was red and thickened. In some the antrum alone, either on one or both sides, was involved and in some the upper nasal cells alone were involved. In all cases, except the one just referred to, improvement in the general condition was noted so quickly after improvement in the nasal condition that

it seemed certain the nasal suppurative process was the causative factor for the kidney condition. This single case just referred to died without improvement.

In one case of antrum suppuration seen first February 5, 1922, the results were most striking. The antra were irrigated on several occasions and quantities of pus were recovered by irrigation. Immediately after this there was rapid loss of edema, and the chemical and microscopic findings in the urine at once began to return to normal. After about ten days there was an increase in the edema and in the amount of albumin and microscopic findings. The antra were irrigated and found to contain pus. Improvement like that noted after the first irrigation again occurred. This treatment was repeated on several occasions within a few weeks with similar results in each instance. This case was a fair example of the results obtained in cases of antrum suppuration. In some cases of suppuration of the upper nasal cells the nose was treated by the instillation into the nose of 1 per cent mercurochrome (220) solution twice daily. There was rather prompt and continued improvement in the suppurative process in the nose. As in the other case the improvement in the general condition was parallel to that in the nose. After the urine had become normal, generally in ten days to two weeks, these children were placed on normal diet without any recurrence, so far, of the kidney condition after the nasal condition had cleared up.

Prior to the institution of nasal treatment they had failed to respond to the treatment usually given these cases, rest, special diet, etc. The following case reports were given:

"Case 1.—A six-year-old child was brought to the office May 27, 1921, complaining of nasal obstruction and sneezing. It was noted that she had a chronic inflammatory process in the sphenoid region on both sides and a sufficient mass of adenoids to cause the nasal obstruction. The adenoids were removed and with local treatment of the nose the inflammatory condition cleared up considerably, although at times she was inclined to have a condition which was suggestive of hay fever.

"On March 27, 1922, a pediatrician asked me to look at her nose to see whether there was any cause there for headaches and fever. There was a history at this time that she had re-

cently had what the family called "flu," which was a moderate epidemic of rather severe nasal infection which was prevalent at the time. It was noticed that her face was slightly swollen and puffy beneath the eyes. There was pus in the middle meatus on both sides and swelling of the middle turbinates. There was heavy golden yellow pus in the nasopharynx, apparently coming from the sphenoid. Urinary examination disclosed a very active acute parenchymatous nephritis. Swelling of the face, legs and abdomen continued until about April 3rd. Since March 27th she had been on daily nasal treatments and rest in bed. On April 7th, her condition was very much improved. There was much less pus in the nose, and it was also noted that her general condition improved coincidentally with the nasal condition. Dr. T. Wistar White felt certain that the kidney condition was caused by the suppurating focus in the nose. She gradually improved and cleared up. Cultures from the nose showed pure staphylococcus albus.

"Case 2.—A six-year-old child was admitted February 5, 1922, to the St. Louis Children's Hospital, with swelling of face, eyes and abdomen. There was no history of any previous serious illness. About the first of December, 1921, he had a severe cold with profuse discharge from the nose, no cough and did not complain of throat. On December 15th, his condition was diagnosed as acute nephritis. The urinary output at this time was 60 cc. per day with albumin 4 plus and casts. He was put on milk diet and for a while the swelling decreased and the urine improved. About February 1, 1922, the patient developed a head cold and sore throat and his mother noticed that his abdomen was swelling again.

"Physical examination on admission showed eyes swollen, abdomen large and patient very listless. Small amount of bloody discharge from right nostril, mucous membrane reddened with strings of mucopus everywhere. The tonsils were large but not acutely inflamed.

"February 6th, there was complete anuria; the next day the output was 150 cc. The nose was treated with aqueous solution of phenol and iodine, and on the following day there was less swelling of eyelids. On February 11th, the patient was much brighter and the output was 650 cc.; edema of the ankles was less. Nasal irrigations three times daily. February 13th,

complete anuria, complained of pain in left ear. February 14th, less pus in the nose, left tympanic membrane bulging. The 15th, the left tympanic membrane opened with free drainage of pus. Edema marked. On the 17th, there was an acute otitis media with free drainage. On the 18th, the urinary output increased. It is certainly evident in this case that edema increased parallel to the infection and that treatment depends upon the removal of the focus.

"On February 21st, he voided approximately 1,000 cc. in twenty-four hours. There was less discharge from the nose and the temperature was normal. No note of change in edema. On February 22nd, the child was much brighter; urinary output, 1,200 cc. Weight, 52 pounds. On the 26th, his temperature went to 104° F., and there was a small white patch on the left posterior pillar. The right tympanic membrane showed a tendency to bulge. K. L., negative. On the 28th, the temperature was 103° F., and there was considerable amount of pus in the nose, which was apparently coming from both antra. The following day the temperature remained the same, and there was marked ascites with scrotal edema. On March 7, 1922, the temperature was 105° F. A trocar was inserted under gas anesthetic and considerable pus removed from the left antrum by irrigation. On the 10th, there was a loss of three pounds in weight and the temperature was down to normal. On the 14th, there was very little pus in the nose. On the 16th, the temperature was normal. The nose had cleared up and the edema was gone. The urine still showed a large amount of albumin, a few casts and no blood. On the 17th, there was a considerable amount of pus in the nose. The eyelids were puffy. On the 20th, there was not so much pus. Both antra were irrigated and culture showed staphylococcus. On the 25th, there was a gain of three pounds; slight puffiness of the face, and abdomen seems a little larger. The antra were probably filled with pus again. On the 27th, there was considerably more pus in both middle meati than was seen on the previous examination. Considerable mucus was removed from each antrum on irrigation. On April 4th, the patient looked distinctly more edematous. Both antra irrigated and a large amount of pus recovered from both. Cultures show staphylococcus albus. On the 10th, there was less pus in the nose and

he had lost a pound and a half. On the 13th, the weight went up again to 50.5 pounds. The face was very edematous and the eyes closed. There was free fluid in the abdomen, nose was swollen and completely stopped up. Both antra irrigated. On the 15th, the patient looked brighter, less edema, nose clearer. On the 18th, the patient was very much better, nose clear and free from pus. On the 20th, the urine was still strongly positive for albumin and casts, had lost weight steadily for the last four days, now 47.9 pounds. Edema and general condition much better. On the 25th, he still had considerable secretion in nose. Culture showed staphylococcus aureus, a gram negative coccus and a gram positive bacillus. On May 3rd, the antra were irrigated and there was very much pus, the weight increased and there were signs of edema in face and legs. Many casts in urine and strong albumin reaction. On the 9th, his nose was full of pus, right antrum irrigated and large quantities of heavy pus returned. On the 12th, there was very little pus seen in either side, nose much cleaner.

"Discharge Note.—The persistence of albumin and periodic rise in temperature accompanied by return of edema made us quite certain that there was a focus of infection which kept up the nephritis. It was found to be in the sinuses, especially the antra, and these were repeatedly punctured and irrigated. It was a striking fact that as the pus accumulated in the nose and the temperature became elevated the edema would increase. The patient was discharged May 13th, under protest, after having been in the hospital for three months. At that time there was no edema and the nose was free from pus. He was on a liberal diet with only salt restricted. The maximum N. P. N. was 45 and the minimum 40.

"We have heard from the patient within the last month and he apparently is getting on very well.

"Case 3.—A four-year-old girl was admitted to the St. Louis Children's Hospital on September 20, 1922, with a complaint of kidney trouble. She gave a history of having had whooping cough, chickenpox and influenza. She is subject to frequent long lasting colds, accompanied by cough and sore throat.

"Last June she went in swimming and a few days later her mother noticed a swelling in her legs and eyelids. The urine

at that time was scanty and loaded with albumin. She was treated at home with various dietary measures.

"On admission physical examination showed a well developed pasty complexioned little girl with general anasarca. Her weight was 20.5 kilos, the urine contained a very large amount of albumin, hyalin casts and the output was markedly low. The tonsils were large and cryptic, no exudate. The cervical glands were somewhat enlarged on both sides.

"Adenoidectomy and tonsillectomy on September 22, 1922; adenoids were found to be quite large. Following operation edema of the face and limbs became even more marked. The urine still contained heavy traces of albumin.

"On October 6, 1922, when I first saw her, the right side of her nose was filled with pus and crusts of the same character as we had noted in two other cases of a similar nephritic condition. The antra were irrigated and were apparently clear. There was a stream of pus coming down from the left middle turbinate—i. e., from the upper nasal cells. Cultures showed staphylococcus albus. Nose was ordered sprayed twice daily with 1 per cent mercurochrome.

"On October 7, 1922, the weight was 20.15 kilos, the output of urine was 75 cc., the albumin was plus 4, flooded with hyalin casts, a few erythrocytes and many white blood cells. The edema was so marked that her eyes were practically closed, and the skin over the abdomen and legs was very tense and shiny. Her diet was very restricted and was entirely salt free. This statement covers the situation back as far as September 26, 1922; in fact, her diet had been so restricted since September 20th. She also had previously been treated along similar general lines from a dietetic standpoint without any definite improvement.

"On October 8th, the urinary output was 150 cc., and weight was 20.09 kilos. On October 9th, output was 570 cc., weight 19.8 kilos, and the left thigh appeared to be smaller. On October 12th, the output was 1,300 cc., and the weight 13.5. During this period a treatment was adhered to of strict diet, salt free, and local spray of nose with 1 per cent mercurochrome twice daily. The urinary output improved greatly, there was less albumin, no casts and no white blood cells. On the 13th,

there was no albumin and microscopic examination was negative in every way. Urine was considered surprisingly clear, but the nurse declared that the specimen under question was from this patient. There was still some pus in the nose, but it showed a great improvement over the first examination.

"The weight continued to decrease until on the 16th it was 12.72 kilos. On the 15th, Dr. Mettle made a note that the child had made an almost miraculous improvement. In the space of forty-eight hours she had put out a large amount of urine, with a corresponding loss of weight and edema. The edema and ascites had practically disappeared, and the urine was negative, both chemically and microscopically. Diet was ordered increased and a proposed nose operation postponed because of the great improvement. It was apparent at this time that the betterment of the general condition and of the nasal findings was coincident.

"On the 16th, there was very faint trace of albumin, occasional red blood cells and casts. On the 17th, there was still considerable pus in the nose, and microscopic examination of the urine showed two or three hyalin casts per low power field, a few red blood cells and white blood cells.

"On the 18th there was a note that the albumin had increased, and on the 19th the weight was found to be increasing fairly rapidly, together with the increase in albumin. Weight was now 14.54 kilos and the output 450 cc. Pupils were dilated but react to light and accommodation. An increase in edema was noted and the question of possible mercurial irritation of the kidney considered. Use of mercurochrome was ordered stopped for two days. Nasal findings on the 19th showed a considerable stream of heavy, yellow pus issuing from the left sphenoethmoid region, and the posterior wall of the pharynx was covered with pus. No pus was to be seen elsewhere in the nose and throat.

"The patient was about the same on the 20th, albumin slightly increased, with numerous granular casts, but no red blood cells. Weight, 14.5; no output measurement made. On the 21st, there was less albumin, many red blood cells and weight was 14.31. The weight remained practically the same, being 14.4 on the 24th. The urine gradually cleared up so that it

was entirely negative on the 28th. Nose and throat examination on the 28th showed a thin film of pus on the left side above the middle turbinate.

"The child remained in the hospital on regular diet for about two weeks without recurrence. She is growing normally and appears to be in perfect health."

DISCUSSION.

DR. L. W. DEAN was much interested because he had had an opportunity to treat a number of cases of nephritis in infants and young children who had suppurative paranasal sinus disease. His results were just the opposite of those obtained by Dr. Arbuckle. All the cases treated, however, were cases of subacute or chronic nephritis. Dr. Dean has been informed that in Portland, Oregon, the same good results in nephritis in infants and young children as reported by Dr. Arbuckle were secured by treating suppurative paranasal sinus disease.

DR. EDWIN MCGINNIS was much interested because this was confirmatory of some work Dr. Carey had been doing on some interesting cases, the bacteriology of the nasal sinuses and not the pathologic condition. They found the staphylococcus aureus hemolyticus and experimentally could also get a nephritis from an intravenous injection of the bacteria. Recently in the practice of Dr. Gill he had seen a case of staphylococcus aureus antrum infection and cleaning up the antrum eradicated the nephritic condition. He thought that cases of nephritis should be referred to an otolaryngologist to see whether there was a staphylococcus aureus hemolyticus infection in the nose, and whether clearing up this infection would cause a disappearance of the nephritic condition.

DR. MILLARD F. ARBUCKLE, St. Louis (closing), said that giving gas to these children did not seem to injure them in any way. He believed it to be a general toxic condition of all the cells of the body and not of the kidney alone, and that it was the cause of a disturbance of cell tension. Their bacteriologic work was being done but was not complete and consequently he did not wish to mention it.

DR. NORVAL H. PIERCE presented a further report on

"A New Method of Closure of the Eustachian Tube."

DR. PIERCE thought all would agree that it was a consummation devoutly to be wished to have a method whereby the eustachian tubes could be closed with a degree of certainty. He started the work just after the war, where they saw the marvelous things that could be accomplished by means of flaps in the closure of facial injuries. There were two problems to be considered: First, the closure of the tube and, second, the fixation of the flap over the tympanic end of the tube. He started in by knotting a strand of catgut and passing it up the eustachian tube after attaching it to a bougie, the knot being sufficient to hold well in the isthmus, and then it was withdrawn through the nose and cut off and then drawn up again into the nose through the tympanic cavity and cut off there. This proved unsatisfactory because it often did not hold and he then thought of using a flap. The taking of a flap from the posterior surface of the mastoid was an old procedure. It occurred to him that by fixing the catgut in the isthmus, the end projecting through the cavum would be sewed through this flap and by anchoring it with a split shot it could be held in contact with the tubal opening, the tube to be well curetted and the tubal cells well broken down, so that there would be a raw surface to which the flap could adhere.

He had had seven cases and there was no failure so far as he knew. There was one failure in a case he had not seen since shortly after the operation. That case was in a child who had been operated upon previously, and there was a scar and a fistula from which pus was discharging. The child was tuberculous and the operation did not go well. The stitches had to be removed a day or two after the operation from the wound behind the ear, and there was a wound which healed by granulation. When he saw the child, about two weeks after the operation, there was no sloughing, but he had been informed that this had occurred since then. The operation was not so well adapted to cases where a mastoid scar existed. The elevation of the tissues for the flap was much more difficult, and there was no doubt that nutrition of the parts had been so materially decreased that failure might follow by a

sloughing of the flap. One charming young woman had a lock of hair growing out through the meatus as a result of the hair being caught in the flap. The last patient operated by this method was dry in a comparatively short time, and the hearing was very markedly improved. All the cases were carefully tested out as to the cochlea, vestibular apparatus and the stapes, for if there was ankylosis of the stapes no improvement could be expected. In his opinion a carefully performed radical operation increased the hearing for a considerable time. Naturally, the hearing would decrease with advancing years and as the stapes becomes fixed, but this had nothing to do with the radical operation. There had been no relapses in his small series of cases and the tubes had been sealed. Only time could tell whether the operation would be of any great benefit.

(Dr. Pierce then showed a series of lantern slides illustrating the various steps of the operation.)

DISCUSSION.

DR. ALFRED LEWY said the numerous methods devised for closure of the eustachian tube indicated by their number how difficult it was to achieve a reliable closure. According to his observation the several methods of curettage, and skin grafting, whether primary or secondary, seemed to make very little difference; the tube closed or did not close apparently under conditions often beyond the control of the operator, even in cases which had had the most painstaking care of the nose and throat.

He had seen Dr. Pierce perform his operation and there appeared to be nothing technically difficult about it. Dr. Pierce had very ingeniously developed a method of applying the well known pedicle graft for the closure of the eustachian tube and it promised well. The one unsuccessful case mentioned by Dr. Pierce he had seen. The boy was a tubercular subject and had recently been operated by himself for a large subperiosteal abscess, at which time the entire mastoid process was found to be occupied by a cholesteatoma which was removed, but no plastic was done on account of the condition of the soft tissues. At the secondary operation Dr. Pierce undertook the pedicle flap operation but the stitches all sloughed. It was, of course, altogether an unfavorable case for this operation.

The latest book on otology seen by Dr. Lewy, that of Dan McKenzie, who attempts to close the eustachian tube by a primary Thiersch graft, stated that many of his cases discharged for six months, and about 25 per cent continued to discharge indefinitely. He believed that any method that promised definite closure was worthy of serious consideration. The only thing he could think of that would keep Dr. Pierce's operation from universal adoption in properly selected cases would be for Dr. Bárány's operation to prove successful. Dr. Bárány, as they all knew, treated the mastoid cavity and the tympanic cavity as separate entities, and endeavored to obtain separate closures of the mastoid by granulation and then treated the tympanic cavity as necessary. If this technic proved successful, of course the pedicle graft for closure of the eustachian tube would be unnecessary.

DR. GEORGE E. SHAMBAUGH thought that of the numerous suggestions that have been offered for securing a permanent closure of the tube the one offered by Dr. Pierce seemed to assure the best chances of success. He had never been able to get very much excited as to whether the tube remained open or closed after the radical mastoid. In the first place, in those cases where there is a persistence of moisture after this operation, it was only in a few of these where such persistence could be reasonably attributed to the nonclosure of the tube. One should always explain to the patient before undertaking the radical mastoid operation that this operation is not done because of the discharge alone, but it is undertaken in some cases of persistent discharge because in connection with this discharge the examination has convinced one that there exists a dangerous focus of infection, a caries or a cholesteatoma hidden away in the attic, aditus or antrum, and that the operation is undertaken for the elimination of this focus which is recognized as a distinct menace and a possible cause for a serious intracranial complication. As a matter of fact, when the radical operation has been properly carried out and the condition described above which constitutes a menace has been eradicated the object for which the operation was undertaken has been accomplished. Should there persist a moisture in the tympanum because of local conditions here, or because of a patulent eustachian tube, that was no indication that the

object for which the operation had been undertaken had not been attained. It should not be a difficult matter to explain this situation to the patient.

DR. SAMUEL SALINGER expressed his pleasure at having heard the paper and at having seen the pictures, which made plain many refinements in the details which he had not understood before. A short time ago Dr. Wittmaack published an operation very similar to that described by Dr. Pierce, in which he stated that he had performed this operation on 100 cases, in the majority of which he had obtained a splendid result. Dr. Salinger had tried it in two cases at the County Hospital, in both of which the flap had sloughed. He thought this was not due to the operation but to the operator, for he could see that several differences existed in the technic as they had performed the operation and that demonstrated by Dr. Pierce. In one case they did this operation on one side and the flap sloughed; a few weeks later they performed a radical mastoid on the other side and did the Bárány operation with very good results. They have done two subsequent operations by the Bárány method with excellent results. In all the cases they found an incomplete pneumatization and very shallow mastoid cavity, so all they had to take care of was the aditus and tympanic cavity. In both cases the posterior wound seemed to heal up by granulation, and the tympanic cavity derived its covering from the intact meatal covering. He thought in this type of mastoid (sclerosis with few or no cells) any plastic was unnecessary, for by leaving the membranous meatus intact the shallow mastoid cavity was shut off from the meatus, healing by granulation, while the epidermis grew in more rapidly to line the tympanic cavity and tubal opening.

DR. EDWIN MCGINNIS said that at the last meeting of the Clinical Congress of Surgeons held in Boston, Dr. Smythe demonstrated a new radical mastoid procedure. He makes a flap of the posterior portion of the external canal. This could be used to sew into the eustachian tube by the Pierce method. He exhibited four or five cases, and there was no sloughing of the flaps, and the results were excellent.

DR. ROBERT SONNENSCHNEN said he had had the privilege of seeing Dr. Pierce perform one of these operations and that it was a very beautiful performance. While it was true, as

Dr. Shambaugh stated, that it was not absolutely essential to have a perfectly dry ear, it was a great desideratum, and if this or any other method would give a dry ear it would be very pleasant, especially from the patient's standpoint. He thought all would agree that in the performance of any radical mastoid very careful attention to detail was necessary or the result would not be satisfactory. Not only the operative skill which Dr. Pierce possessed but his very careful attention to the details of after-care probably accounted for his success.

DR. CHARLES H. LONG reported a successful closure of the tube by skin grafting, according to the technic of E. Hamilton White of Montreal, which was published in the *Journal of Laryngology and Otology of Edinburgh, Scotland*.

On August 24, 1922, a radical mastoid operation was performed on a male patient fourteen years of age. On September 7th, or fourteen days after the radical operation, an exact cast of the bone cavity was made by pressing into it a piece of modeling compound. When this hardened the cast was removed and a skin graft taken from the arm was laid over it with the cut surface outward and it was then pushed back into position. Seven days later the cast was withdrawn through the retroauricular opening. November 15, 1922, the eustachian tube was found closed and the ear was dry. At no time had the patient experienced any pain or discomfort. Both cases reported by Dr. White had dry ears in less than a month after operation. He made no mention of the condition of the eustachian tube. Further experience was needed before this simple procedure could be recommended as a regular method for closure of the eustachian tube, or for shortening the usual long drawn out after treatment or postoperative mastoids.

DR. HARRY L. POLLOCK thought that so few of the radically operated ears discharged from the tube that it was not necessary to attempt to close the end at the time of the operation. Of course, it was better, but there was only a small percentage that gave any trouble. A few weeks after the radical operation it was very simple to close the pharyngeal end and in those cases where the persistent discharge occurred they closed it up with little trouble in every case simply by pulling out the mucous membrane of the pharyngeal end and clasping it together. In practically every case the eustachian tube closed following

this and there was no further trouble from discharge in the mastoid cavity. If every radically operated ear had this trouble Dr. Pollock thought it would be necessary to close the tube, but the percentage was so small that it was no trouble to close the tube afterward. He had never attempted the operation described by Dr. Pierce, but if it was so simple he believed it should be done at the time of operation rather than four or five months later. He saw no reason why it should not be successful in every case.

DR. NORVAL H. PIERCE (closing) said that Dr. Shambaugh claimed that he was not worried about a discharge after a radical operation. Dr. Shambaugh was not the one to be considered—it was the patient, and the patient worries. The patient regards a running ear after an operation as a failure in the operation. He thought it was true that the factor of a running ear had been eliminated as a fatal issue, but he disagreed with Dr. Pollock that these running ears occurred so infrequently that it was a matter of no importance as to the closure of the tube. The literature gave from 45 to 60 per cent of failures as to running ears after an operation.

It was true that Wittmaack published an operation a little while before Dr. Pierce's was published, but the operations were different to the extent that Wittmaack does not fix his flap as Dr. Pierce fixes his, in the mouth of the eustachian tube, and Dr. Pierce thought this was perhaps the most important part of the technic.

Regarding the operation of Professor Bárány, that was probably done on cases which he would not operate upon at all; cases of arrested pneumatization with a slight discharge from the ear did not require an operation, and where a large cavity existed with a large cholesteatomatous mass this operation was not to be thought of and one did not have to have any experience with Bárány's operation to realize this. That conclusion had been going on for years and years. Schwartze, who inaugurated operations on the mastoids, treated the chronic cases in just about the way Professor Bárány does at present. He made no flap, but treated the wound from back of the ear, and the failures in these cases were so numerous that the radical operation was devised to more frequently insure success. He thought all would agree that if one operation could accomplish

what two would do it was better to do it in one. Dr. Pollock required a second operation with the method mentioned by him, and so also did the method mentioned by Dr. Long, but the one stage operation was surely to be preferred.

DR. GEORGE W. BOOT said that as Dr. Bárány was not present to defend himself, he thought someone of those who took his course should mention the fact that Professor Bárány stated definitely that he performed his operation in cases of cholesteatoma.

DR. PIERCE, replying to Dr. Boot, said he did not wish to assault Professor Bárány. He said, and repeated, that the operation as performed by Professor Bárány was, in his opinion, inapplicable to large cholesteatomatous growths in the mastoid.

DR. AUSTIN A. HAYDEN presented a paper entitled:
"Fifth Year Oto-Laryngology in General Hospitals."
(Preliminary Presentation.)

ABSTRACT.

The scope of this paper was limited to the consideration of otolaryngology in the graduate, hospital or fifth year medicine. Postgraduate teaching is entirely another matter which Phillips, Shambaugh and others have dealt with ably and thoroughly. The term "General Hospitals" was meant to include the typical average better grade institution of 100 beds or more with rotation interne service, the men serving successively for given periods of time in the laboratory, obstetrical, medical and surgical departments. This excluded special hospitals and those with single nonrotating house services, where a whole year is spent in one department, as well as those having a special eye, ear, nose and throat interne while the rest of the house staff rotates.

The teaching in diseases of the ear, nose and throat should properly come from the attending otolaryngologists.

The fifth year requirement, inaugurated a little more than a decade ago and still largely in the formative period, had in reality created a new national American medical school whose field is just above the undergraduate and just below any sort of postgraduate instruction or the years of practice that are to follow. It is an in between period in which the student is no longer a student on the benches, nor is he yet a full fledged

Doctor of Medicine. The student body numbered 2,529 last year. It would be considerably larger in 1922-23. The average age was somewhat over 27. The per capita investment had been variously estimated as between \$10,000 and \$20,000, or from \$20,000,000 to \$40,000,000 in all.

Dr. Hayden said that to present a complete survey of this whole subject would be manifestly impossible and entirely inadvisable, and after analyzing the actual otolaryngologic work done in a 166 bed hospital, with which he was familiar, he gave the following conclusions:

"1. Otolaryngology forms a very considerable part of the work of the average general hospital. For that reason internes are entitled to a proportionate amount of training in this department.

"2. A curriculum carefully worked out by otolaryngologists and followed by them in their own hospitals would equip the general practitioners of the future with a broader and more practical knowledge of the diseases of the ear, nose and throat.

"3. From these more systematically trained physicians undoubtedly adequate numbers would be attracted to this specialty after having spent a number of years in general practice or suitable postgraduate work.

"4. If advantage is not taken of this opportunity otolaryngology will gradually but surely lose its identity as a specialty and be merged into general surgery or general practice.

"5. A standardized curriculum for systematic fifth year instruction in all departments of general hospitals might prove to be as long a step forward in the further development of modern medicine as was the standardization of the medical college itself. Otolaryngology, if it will but lead the way, can render great and lasting service to organized medicine."

DISCUSSION.

DR. GEORGE E. SHAMBAUGH said that he considered the subject presented by Dr. Hayden as an extremely timely one. We need more discussions of subjects of this type.

The primary question brought up was this: What should be attempted in the general hospitals along the line of educating the internes in otolaryngology? Dr. Shambaugh felt that the solution of this question was not difficult, especially for

those who have been interested in the subject of instruction in otolaryngology for undergraduates. This point should be kept clearly in mind, namely, that the fifth or hospital year should not be looked upon as a graduate year, but as a part of the undergraduate medical training and as intended to round out the clinical experience of the undergraduate student where the work in the previous years had, in a sense, fallen down. When planning the work for the fifth year in hospitals this fundamental fact should be kept in mind, that the object of undergraduate work in medical training is to prepare men to be general practitioners. There is no place for specialization in undergraduate training. This should apply as much to the hospital year as to the previous years' work. Those who are most interested in medical education are at one in the opinion that the training for undergraduates in the various special fields should not include training in operative technic. For example, in otolaryngology the aim should be to train the undergraduate medical student how to carry out the simpler examinations; also to present to him the various clinical problems encountered in this special field, but it is a mistake to try to teach him surgery in the ear, nose and throat. He was inclined to believe that the general surgeon has been slower to appreciate this fundamental fact in undergraduate medical training. He believed that what is required as applied to the operations on the ear, nose and throat applied in equal force to general surgery. The principles of either one of these subjects and the problems encountered in practice are proper subjects for introduction in the undergraduate curriculum. Operative courses and surgical clinics should be largely dispensed with. These should form an important part of graduate training for those who are going into special practice; whether this be otolaryngology or general surgery, it is one and the same thing.

Regarding the field of otolaryngology, Dr. Shambaugh felt that a great deal of harm is being done by efforts to teach the general medical student the technic of operations either in the college years or in the fifth year. One should always bear in mind that it is a relatively simple matter to teach a man how to do an operation, but it is much more difficult to teach him to recognize when to do these operations. It has become the practice in some general hospitals to allow the internes in their

regular fifth year's work to do operations in the nose and throat, the idea being that these are minor procedures and that the technic can be readily mastered. It is, for example, a simple matter to teach a person to do a turbinectomy, but anyone trained in otolaryngology recognizes that it takes many, many months of constant study of clinical patients to instill into the student's mind an appreciation of the proper indications for a turbinectomy. All this applied just as truly to the question of adenoids and tonsils. To train general practitioners in the technic of operations in this special field was only preparing men to increase the flood of unnecessary operations on the nose and throat, and particularly upon the tonsils, that has been flooding the country in recent years.

Dr. Shambaugh stated that he was very much interested in training men in otolaryngology, but he did not approve of teaching general practitioners the operative work before he had some assurance that they had studied the subject long enough to understand the indications for operations.

DR. JOHN MILTON DODSON found himself thoroughly in accord with Dr. Shambaugh about the limitations of the field and agreed heartily with him that the intern year was not a graduate year at all. One of the reasons given for making it a requirement for graduation was to emphasize that very fact. He thought all would agree that no young man could safely be trusted to go into independent practice before having practiced for a considerable period under the immediate supervision of an older, more experienced man. That experience he obtained in the older days under the preceptor system. When the preceptor plan, for various reasons, fell into disuse he could obtain this training only in a hospital, but there was a considerable period between the time of the preceptor era and the development of a sufficient number of hospitals, so that, during this interval, a large proportion of the graduates in medicine went into general practice and secured their training and experience on the patients who came under their care. Some twenty years ago the practice of medicine began to be hospitalized and these institutions began to increase rapidly in number. The interne year was not new. For fifteen years the majority of students have sought to secure and serve in-terneships. The few schools that have taken the step in the last

five or six years to make it a requirement for graduation have done this in order to get the interne year under supervision of the faculty so that they may advise them and assist them in the selection of internships and thus get some control over the work they do in the hospitals and, secondly, supervise this phase of the hospital's work. The great purpose of the movement is to make the hospitals do better work, have better supervision over the interns and realize their responsibility to the internes.

Dr. Dodson thought Dr. Hayden's figures were rather excessive, that the average expenditure of medical students would not be as high as he indicated, but they had invested enough. They had reached the age of twenty-seven, in most instances, when they entered the hospital to give hard service for a year to fit themselves for the practice of medicine and they were entitled to close supervision and all the assistance the members of the staff could give. The only way to get good and efficient service was to give the necessary time to the internes.

As to the number of internes required, he thought Dr. Hayden's figures were far too small. The 428 hospitals that had been approved by the Council on Medical Education of the A. M. A. by no means represented the thousands of hospitals in the country that needed internes and were entitled to them. If all the hospitals which could provide reasonable opportunities were to demand internes in the right proportion—not over twenty to twenty-five beds to the interne, especially on the medical side—the number of internes would not be anywhere near sufficient. The acute problem was how to provide resident services for all of the hospitals, which are rapidly increasing in number. The number of graduates was not going to increase and it was only about 3,000 a year.

When asked what time shall be given to otolaryngology in the fifth year the question arose, What are we making of them—specialists? He hoped not. They were trying to make the good old family doctor, who was said to be passing but without whom he did not believe the world could get along. Every medical man should know some otolaryngology as related to medicine, and he still thought they could best be taught this by an otolaryngologist, but was beginning to question this a

little. If the interne saw the medical man in the hospital needing the assistance of all these specialists, how was he ever going to get along in the country, where he must examine the ears and the nose and the eyes of his patients and have at least some degree of experience in the special lines? Dr. Dodson thought the otolaryngologist should do a certain amount of teaching, but he should bear in mind constantly that so far from attempting to teach the technic of the operations to the young men he should be constantly emphasizing the fact that they should not attempt many of these operations. It was not a question of knowing how to operate but when to operate. He believed no man should hold himself out to the public as fit to undertake these operations until he had been at least five years in general practice, and then had spent at least one, preferably two, years under favorable conditions for special training in that particular line. This seemed a long road, but the rewards would be much more in proportion, if one could judge from the fees charged, than the fees the country doctor got for the preparation he had to make.

Dr. Dodson thought it was hard to see how the students could be taught much otolaryngology in the fifth or hospital year. One must remember in the fixed services that the hospital, even a teaching hospital, has obligations other than those to the patient. The hospital is first of all for the care of the sick, and no other phase of its activity can be permitted for a moment to prejudice in any way the welfare of the patient. There was no question that for the patient's interest and for the comfort of the attending man the service of a young man for a whole year in one department—e. g., medicine—is much better than service in a department for only two or three months. Under this latter rotation plan, he is just beginning to get fit when he is shifted to something else. The young man should follow a year by a year or half year in surgery; it made little difference whether in special or general surgery, but he should learn the principles of surgery. Even in the rotating service Dr. Dodson could not see how anyone could teach much otolaryngology unless the student was restricted entirely to that service, as he is at the County Hospital for three months. At present a man may finish his County Hospital service without any work at all in otolaryngology or some

other of the specialties, but Dr. Dodson thought this not a serious matter.

What was the solution? He believed it was in the very beginning of the organization of the interne year and thought the time was coming soon when hospital staffs must be organized into an intelligent teaching unit. Not that they should organize into a lecture bureau and harangue the students, for they had had enough of that in the medical school, but he thought it should be possible to so arrange the work that an interne on the medical service should have an opportunity to learn all he needed to learn in otolaryngology and ophthalmology while serving his year as a medical interne. He was not quite clear as to just how this could be brought about, but felt that a young man who served after four years of medical training, in residence, a year or two of internship in a hospital should go out with a good training in some one line and a reasonably clear knowledge of the special branches of medicine, and that the staffs of the several hospitals where internes go should be alert about this matter, should recognize and give study to the pedagogics of the interne year, just as the faculty of the undergraduate school must do in order to get good results.

DR. J. HOLINGER said he had been connected with internes in general hospitals for more than twenty-five years, and his experience was that the question how much an interne learned and how much he missed was largely personal. Some internes took to the work like a duck to water. They went into it with a will and were glad of an opportunity to learn. They would take patients into the examining room, examine them and after a time come back with questions, and it was a pleasure to teach them. By far the largest number try to dodge such work and do as little as possible. He believed that if the colleges expected the hospital staffs to do something for these boys they should give the members of the staffs the authority of making the boys do their share. Many internes acted as though they did you a favor when they listened to your teachings. If an interne became too selfconscious and overbearing, Dr. Holinger asked him to examine a patient and tell what he found. Some recognized their shortcomings; others would find some excuse to get away. In his opinion, all students should have

an idea of the technic of a general examination of the ears, nose and throat. This they were entitled to. They should know a drumhead when they see it, the main parts of the nose; they must know how to handle a head mirror properly, and they should be able to look into a mouth and pharynx without making the patient vomit. Dr. Holinger always found that it was an advantage for the student to have his own forehead mirror and ear and nose specula; then they will use them and learn something. The student should recognize that he is under the supervision of the staff and give as much assistance as possible.

DR. ROBERT SONNENSCHNEN thought one of the crucial facts was that in the past few years there apparently had been a different method of specializing, at least so far as time was concerned. In former years the men who were recognized as specialists were those who, after leaving the hospital in which they had served an internship, had taken a course in some special branch or had worked with a man doing such work on the living or the cadaver before proclaiming themselves as specialists. This took from two to five years, and many of these men had practiced general medicine for several years, thereby fitting themselves better to specialize in some particular branch. In the last four or five years a great many of the younger men had specialized immediately upon leaving the hospital. He had the honor of being connected with a hospital with rotating services, and if one examined the operating room schedule at that hospital it would be seen that an enormous percentage of the operations being performed were by men other than specialists or by very recent exinternes and graduates who came in and did turbinectomies, tonsillectomies, etc. In this hospital, and probably in many others, in order to get any service from internes it was necessary to give them practically all the operations on the ward cases. If through the kindness of the men on the staff the internes could be taught the technic of the operations they may have to do if they expect to do country practice, or if they intend to take up a specialty, it was all very well, but, on the other hand, it was a question whether all the operations must be turned over to the internes. He believed that on a rotating service of three months the attending man could teach the functional tests of

the ear, the examination and even the operating in the parts assigned to otolaryngology.

Dr. Sonnenschein believed that the only cure for this generalized operating by internes or any group of them would be for the general hospitals that have a rotating service to have an agreement regarding the operating to be done by the internes. There would then be no advantage in going to any particular hospital merely in order to get plenty of operating whether the man showed by his attention to duties that he deserved the operative work or not.

DR. EDWIN MCGINNIS thought the subject was very important and said that as he had no regular assistant he had worked out a scheme of having a hospital interne act somewhat as an assistant. He had no special anesthetist, so his interne gave his anesthetics for him. He tried to teach these men how to use a local anesthetic and to anesthetize the throat properly so that they might become competent in examining patients and could handle the patient afterward, trying to teach them all the complications that might arise following an operation. He thought it was difficult to do much for the interne so far as the nose was concerned. One could show him the condition of the nose before operation and after completing the operation could show him the picture so that he could get an idea of the "before and after taking" condition. He thought it was surprising how much help could be obtained from internes in this way. He seldom had to go to the hospital to stop postoperative hemorrhage, for his internes were taught how to get the patient to the operating room and could usually control the hemorrhage without difficulty. In his experience with fifteen or sixteen internes not one of them had expressed their preference for otolaryngology. In the last few weeks three young men had come to him and asked how to go about becoming specialists in otolaryngology. He had told them that they should take a general hospital internship and follow this with a postgraduate course at Rush Medical College and then an internship in a hospital where they could do this special work. This would take two or three years and then if they wanted to go into otolaryngology they would be prepared. Dr. McGinnis had rather drifted into otolaryngol-

ogy through his association with Dr. Ingals, after several years in general practice.

DR. L. W. DEAN, Iowa City, said that of his five internes one had had a service in internal medicine; each of the others had had a rotary service. Each of the four internes who had had a rotary service had operated on many cases for the removal of tonsils. They felt that they could do the work well when, as a matter of fact, they were not capable of properly performing the operation. The one interne who had a service in which he had no tonsil work was very much discouraged at first because he felt the other internes were more proficient than he. He was advised that as he had built his foundation well, he was much better prepared for the work than the others. Dr. Dean believed that the performance of operative work by internes before they have been properly prepared for this work could only be prevented by the education of the members of the clinical staffs of the hospitals or perhaps by bringing pressure to bear upon the hospitals to see that these things were done correctly.

A physician practicing in a small town must be able to diagnose suppuration of the middle ear, a diphtheritic or nondiphtheritic throat; he must be able to treat acute laryngitis in the infant, paratonsillar abscess, and many other common conditions. He should know all about the application of various drugs in emergency cases. In order to educate internes so that they get those things which they will actually need in private practice is a problem which can only be solved by some person or persons giving to the internes a lot of time and attention.

DR. GEORGE W. BOOT thought it was impossible to take any one part of the body and separate it from the rest, and that one could not take any part of a hospital and separate it from the other. According to Dr. Hayden's figures, about one-fourth of the hospital cases belonged to the ear, nose and throat department, and the interne taking a general service should be entitled to some knowledge of ear, nose and throat work. The staff was divided into the different specialties, internal medicine, surgery, obstetrics, otolaryngology and so on. In the month of March he had listed eighty-seven medical cases and seventy-seven ear, nose and throat cases, so it seemed

that the interne was entitled to almost as much instruction in otolaryngology as in medicine. Another feature was the limiting of the ear, nose and throat work to specialists. He thought that all medical work was not limited to internists or all surgical work to surgeons.

Still another feature was the fact that thirty-one of the fifty-nine ear, nose and throat operations in March were done by general practitioners, over one-half the work, and Dr. Boot thought these men were going to do this work anyway, and since this was the case they should be shown how to do it, out of consideration for the patient.

DR. NORVAL H. PIERCE expressed the opinion that the interne body was getting very cocky. The internes at a certain Chicago hospital recently held an indignation meeting and demanded as one of their "constitutional rights" that they be given all the tonsil operations. Internes are a necessity. The staff met with them and talked as kindly as possible to them, but it was a drawn battle, and the internes were now deliberating as to whether to continue to serve the hospital or leave the staff in the lurch. They have quite well founded ideas, and among them is the idea that they shall take out tonsils. Dr. Pierce thought that the legitimate indications for a tonsillectomy could be easily acquired. The mere removal of the tonsils did not constitute the main part of the job; it was laying the foundation in an individual of a certain moral standard, and that was a very difficult thing. It was making a man who has a large family and very little money, rather than break through that moral code, tell a patient who did not need to have his tonsils out, "No, you do not need to have them removed," and that was a very difficult thing to accomplish. It probably went even back of the college education or was, perhaps, prenatal. He confessed that he was ashamed of his educational neglect of the internes in the hospital he attended. There was no training of internes. He agreed with the statement of Dr. Dodson that the first requisite of the hospital was the care of the patient. Anything that came in conflict with that was wrong, but they must face the situation of education, not only of nose, throat and ear internes, but the whole interne body. He was inclined to think that within a few years the whole aspect of the thing would change; that there would be

paid trained residents in the better hospitals, which, of course, would give a much better basis for education than the present status of the interne, who was not paid and not appreciated.

DR. JOHN MILTON DODSON agreed that the vital thing to teach any interne was the proper examination of the patients. One of the serious mistakes under the present system was the way patients were assigned. The hospitals and dispensaries were doing the very thing they criticised the general public for doing, letting the patients make their own diagnosis. The patient goes to the hospital and assigns himself—says he has bad tonsils, or bad eyes, and wants to go to that department. They hope soon to have every patient that comes to the dispensary go to the department of general medicine first for a group study and diagnosis as they do in the Massachusetts General Hospital. That is where they belong, because each patient is entitled to know whether he has anything else wrong with him besides what he thinks he has. The interne should not be permitted to report the examination of any case until he has examined the nose, the tonsils, the ear and every other part of the body.

As to the cockiness of the internes, Dr. Dodson thought that was a perfectly obvious result of the demand exceeding the supply and that the only way to overcome it was to make the fifth year a part of graduation, for the interne who does not complete his contract, who does not serve the men on the staff of his hospital, and it is so reported, does not get his diploma and he knows it. On the other hand, most of the faults of the internes and the reports on them are due to the men on the staff. Dr. Dodson did not know any young men who were not anxious to take advantage of any good instruction that they could get. He always told the students that they were not entitled to do any operating unless the attending men thought they should.

DR. AUSTIN A. HAYDEN (closing) expressed his gratification of the general discussion which the preliminary presentation had brought out. He thought Dr. Dodson had misunderstood him as meaning to develop specialists out of the general hospital service. That was not the intent of the paper.

He believed men who were not working in a general open and closed staff hospital did not realize the limitation of such

a hospital. The vast majority of the hospitals in the United States today are such institutions as he had described. There were only two hospitals in Chicago that had the single, non-rotation service, the Presbyterian and St. Luke's. These men are going out with an unlimited license to practice any sort of medicine that their patients bring to them to take care of. He remembered an interne whose first operation was a Cesa-rean section. He believed the first thing the average young man would be called upon to do would be to take care of an obstetric case. They could do this much better for the patient if they had been properly trained in obstetrics by an efficient obstetrician. By the same token, he believed that any interne was soon going to take out some tonsils, probably not quite so soon if he located in Chicago, although Dr. Hayden did not believe that the practice of medicine in Chicago differed intrinsically from that in the rural districts. He felt that if the interne had been as well grounded in the hospital as time would permit by such men as constitute the membership of the Chicago Laryngological Society he would do much less harm to the patients who came to him in private practice.

In his opinion the day of the old family practitioner had entirely passed, or at least he had been supplanted by a new variety of medical man. He remembered the equipment his father had in Southern Wisconsin. There were few nose and throat instruments and the obstetric forceps had a much more prominent place. Conditions have changed, and the man who goes out today very soon develops into an all-round specialist as well as general practitioner. The training that could be given when carefully supervised was something that the interne had a tendency to feel that he had a lien on and that he was really entitled to that teaching, inasmuch as otolaryngology constitutes a very considerable portion of the work of any well balanced general hospital.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, January 8, 1923.

THE PRESIDENT, DR. CHARLES H. LONG, IN THE CHAIR.

DR. CHARLES H. LONG exhibited a patient with

Hare-Lip and Cleft Palate.

The patient was a Norwegian man, aged 34 years, who was born with a cleft palate and harelip. He had had nasal obstruction since birth. The family history was negative. He was one of seven children, three of whom were dead.

The point of interest was the large amount of lymphatic tissue which could be seen in the nasopharynx and nose and the advisability of doing any operation. He had been operated upon for harelip when a baby. His mentality was that of a boy of fifteen but was said to be improving. Dr. Earl Thomas, assistant to Dr. T. W. Brophy, had examined him and expressed the opinion that no operation would be of value at this time. He believed that such apparatus as a dentist could manufacture would do all that could be accomplished.

DISCUSSION.

DR. ELMER KENYON stated that Gutzmann had studied cases with adenoids in connection with cleft palate and had decided that unless there was an extremely good reason for removing the adenoids they should be left alone. If they are removed the voice is invariably made worse, as the adenoids serve as a sort of obturator. Gutzmann thought not much could be said in favor of removing the adenoids, except in cases where breathing space was necessary, when a partial adenoidectomy was the proper procedure. He argued that in the cleft palate class of cases, in which the middle ear was always prone to infection, even with a free nasopharynx, there was not the same imperative reason as exists in cases with a normal palate for removing the adenoids to protect the ear.

DR. LONG (closing) stated that Dr. Sher expected to remove enough of the tissue to give the patient nasal breathing space and that he would then turn him over to the dentist to have the proper apparatus constructed.

Foreign Bodies Removed From the Bronchi and Esophagus.

DR. GEORGE W. BOOT presented some foreign bodies which had been removed from the bronchi and esophagus of various patients. The first was a piece of bone which had been impacted in the throat of a Scotchman who had swallowed it while eating mutton broth. Great difficulty was experienced in dislodging it.

The second specimen was a peanut which a colored boy of three had inhaled and which had been removed from the right bronchus.

The third specimen was another peanut which had been removed from the right bronchus of a boy of three. This was the only patient Dr. Boot had ever had who had died from bronchoscopy. The peanut, which was too large to be removed through the tube, broke in two as it was passing the vocal cords and before he could secure another hold on it the boy choked to death.

The fourth specimen was a tack which had been imbedded in the left bronchus of a baby eleven months old for six weeks.

The fifth was a black headed steel pin, 4 cm. long, which had been inhaled and had shifted to various places. The first picture showed it in the right bronchus. The following day X-ray examination showed the pin in the trachea and the next day in the right bronchus. Every time the boy coughed the pin changed position. When extraction was finally attempted the pin was in the trachea and the boy coughed it out as soon as the bronchoscope was introduced between the vocal cords.

Paper: "The Structure and Function of the Crista Ampullaris."*

BY GEORGE E. SHAMBAUGH, M. D.,

CHICAGO.

*See page 443.

DISCUSSION.

DR. J. HOLINGER expressed his thanks to Dr. Shambaugh for his clear presentation of the function of the crista ampullaris and said the intention of physiology has been from the beginning to reduce the complicated functions of the body to simple laws of physics and chemistry. If we did not succeed in this it was either because we did not understand the functions or because we did not know the laws of physics and chemistry which governed these functions. Therefore recourse

was often had to complicated and undigestible theories. He thought very few people could understand Helmholtz's book. The mathematicians say it contains too much physics, and the physicists say that it contains too much mathematics. Siebenmann and others have succeeded in explaining to a great extent the function of the cochlea. Bárány, Jones, Shambaugh, Ewald and others did the same for the vestibule and semi-circular canals.

DR. ALFRED LEWY said that Dr. Shambaugh's work and ideas had greatly strengthened the arguments in favor of endolymph flow as the causative factor in labyrinthine nystagmus and allied phenomena, but his theory that the duration of the induced phenomena was dependent upon the duration of endolymph flow failed to explain several things. According to Bárány, after-nystagmus may last anywhere from three or four to sixty seconds or more in normal individuals. It hardly seemed possible that the anatomicophysical conditions that are the causative factors of endolymph flow can vary so widely in normal human beings. There certainly must be a powerful neurologic element. One must consider the varying sensitivity and interaction of nerve cells. Especially unexplained is the fact that in the same individual, under the same stimulation, on different days, or even on the same day, with the question of fatigue eliminated, the duration of nystagmus, and especially of vertigo and nausea, may vary. Nor could it be explained in all cases by the development of inhibitory impulses, as seen in trained aviators and whirling dervishes. As a working hypothesis Dr. Lewy thought Dr. Shambaugh had presented the case very strongly, but one must keep in mind that other elements, largely neurologic, beside end organ stimulation by endolymph movement, enter into it.

Dr. Lewy also wished Dr. Shambaugh to tell something of muscle tonus in people with congenital absence of or destruction of both labyrinths.

DR. ROBERT SONNENSCHNEN said that aside from the fact that Dr. Shambaugh always seemed to have a very lucid way of demonstrating anything, a very important fact to be remembered was that it was the careful study of the anatomy of a structure that would enable one to understand the physiology. He remembered the wonderful specimens produced by Dr.

Shambaugh when he first did work out at the University of Chicago, many of which had been exhibited at the Congress of Budapesth, where they had been much admired and complimented.

One point not mentioned in this discussion but which he had heard Dr. Shambaugh speak of several times, particularly when in Washington at the meeting of the American Otological Association, was the point against the Helmholtz basillar membrane resonance theory as regards the large blood vessel which lies just below this membrane. Since it is known in physics that a resonator attuned to a certain pitch always acts for that tone, one can assume that the basillar membrane, if it is the resonator, cannot act uniformly, since the large blood vessel situated just beneath it would vary in its lumen according to the amount of blood supply running through it and the force of the heart beat.

Dr. Sonnenschein again emphasized the fact that the important thing in addition to those mentioned was that a careful study of the anatomy of any part was essential to the study of the histology thereof, and thereby often its physiology.

DR. GEORGE W. BOOT thought that without any doubt tonus originates in the labyrinth, but he believed there was considerable doubt that it originated solely in the crista. There are three different places where it may originate, the crista, the macula and the organ of Corti. These three end organs are stimulated by different motions. The organ of Corti by stimulation of vibrations in the endolymph, the crista by currents in the endolymph and the macula by movements of the crystals in the otolithic membrane due to their inertia. If the crista is stimulated by pressure, one should be able to produce nystagmus if the canal is blocked off distal to the crista and the patient rotated, for pressure would be produced on the cupola, even though the endolymph could not move. He thought this experiment had never been tried. If the tonus was the result of the to and fro movement of the blood in the vessel, the question was the same as in the eyeball. In the eye one could watch the vessels with the ophthalmoscope and see the vessels under considerable magnification. There is absolutely no movement in the vessels with circulation under normal conditions. He thought the theory that there is to and fro movement of the

endolymph and perilymph with the pulse was not tenable, for if it occurs one should be able to see it in the eyes, where one can see and study the circulation under magnification in an organ less rigid than the labyrinth and where to and fro motion should occur more easily.

Another point was in relation to the duration of the nystagmus. Dr. Shambaugh thought the current was kept up longer because of endolymph in the utricle. Dr. Boot thought this theory was not plausible. If a vessel, a pail, is filled with water and a hole made in the bottom of the pail, the water comes out with a force that has no connection with the amount of water in the vessel but solely with the height of the water.

If a vessel shaped like a semicircular canal and utricle be filled with liquid and rotated so as to set up a current in the canal and then stopped suddenly, there will be no variation in the amount of current set up in the canal with different sizes of vessels.

DR. FRANK J. NOVAK asked whether the distribution of the nerve fibers in the crista ampullaris had been determined histologically and what the histologic structure of the cupola was. Also what was the effect, if any, of vascular hypertension and various heart lesions, such as aortic regurgitation, or labyrinthine tonus.

DR. GEORGE E. SHAMBAUGH (closing) said that any one of the questions asked open up a field for possible endless discussion pro and con. He believed that the explanation which accounts most readily for the phenomena was the nearest to the proper solution. In order to get anywhere with such problems it is most important to keep our explanations as simple as possible, for only in this way can progress be made.

Dr. Lewy's questions were very pertinent, and Dr. Shambaugh was sure that the duration of the nystagmus, as compared with the duration of the endolymph current, is sometimes modified by outside nervous conditions. This is especially true in pathologic conditions.

Regarding the disturbance caused by destruction of tonus, the sudden destruction of one ear produces a profound disturbance of equilibrium; a nystagmus develops with the slow component to the diseased side. This disturbance quickly disappears. What happens? First, we must remember that tonus

to skeletal muscles is not all of labyrinthine origin. Extralabyrinth tonus is an important factor. All of these tonus impulses pass through Deiter's nucleus. When the labyrinth is destroyed there is a complete blotting out, not only of the labyrinth tonus on this side, but by what is spoken of as diachisis, there is a temporary depression of the extralabyrinth tonus; within a short time this depression from diachisis disappears and there is left only the loss of the labyrinth tonus. This is adjusted in one of two ways, probably both acting together. In the first place, it seems likely that there may be an increase in the extralabyrinth tonus to take the place of the impulses to the destroyed ear and restore balance. On the other hand, there is ample evidence that the tonus from the remaining ear, which supplies impulses to both groups of muscles, those causing movements toward the same side with the stronger impulses to those muscles causing movements toward the opposite side—after one ear has been destroyed for some time a readjustment takes place, so that equal tonus impulses emanate to both groups of muscles from the normal ear. This is evidenced by the fact that in such cases a rotation toward the normal side produces a nystagmus lasting just as long as the rotation toward the opposite side. An interesting sidelight is thrown on these cases by observing what takes place when there is a sudden loss of one ear in cases where in earlier years there had been a complete destruction of the opposite ear. Dr. Shambaugh has had an opportunity of observing but two such cases. In neither one did there occur that disturbance of equilibrium, vertigo or nystagmus, which accompanies invariably the sudden loss of one ear when the opposite ear is normal.

Dr. Shambaugh did not understand just what Dr. Boot had in mind in his objection to the statement that in the rotation experiment the movement of the endolymph in the semicircular canals was prolonged by the pressure exerted from the fluid in the much larger utricle which is also affected by the rotation but lacks much of the friction from the walls which retards the flow in the canal itself. This conclusion regarding the effect of the fluid in the utricle as well as in the perilymphatic space of the vestibule Dr. Shambaugh considered in the nature of a self-evident fact.

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XLIX.

FURTHER OBSERVATIONS OF MASTOID STRUCTURE BY MEANS OF THE X-RAY.*

BY NOLTON BIGELOW, M. D., AND ISAAC GERBER, M. D.,
PROVIDENCE, R. I.

PREFACE.

Several years ago we undertook some X-ray investigations of the mastoid, the results of which were published in two papers.^{1 2} In these, we attempted to correlate the anatomic data obtainable by mastoid radiography with the clinical and surgical aspects of mastoid disease. As these preliminary studies were limited to clinically observed cases, it became evident that certain anatomic features could be more accurately determined and perhaps standardized by X-raying a large number of bones. In casting about for material, we naturally turned to Dr. B. Alex. Randall, whose collection of temporal bones is world famous. On requesting the privilege of radio-

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Providence, R. I., January 27, 1923.

graphing these bones, we learned that Drs. Joseph C. Beck and Samuel Iglauer had previously made a similar request. At the suggestion of Dr. Randall, it was decided that we all join forces, we ourselves undertaking the mechanical work of making the radiograms. This we have been doing for the past two years, but as the work is more time consuming than first anticipated it has made slow progress and even now is in nowise completed. While experimenting with various angles for projecting the mastoid on to the X-ray plate, we discovered an angle that revealed the cellular structure of the mastoid proper as did no other. This, which we have termed the occipital projection, is equally applicable to the living and now constitutes a part of our routine examination, being especially useful where the cell structure is limited as is the case in infants and young children. We have also found this projection most useful in correlating X-ray appearances with the modern ideas of mastoid development and structural variations so carefully worked out by Wittmaack.³ As our work along this line is still incomplete, it was deemed advisable to present the new projection together with its practical applications as a preliminary report, this with the hearty assent of Drs. Randall, Beck and Iglauer.

It seemed desirable, however, to divide this into two parts, the one discussing the more strictly X-ray aspects while the other deals with anatomic and clinical correlations. Hence, those desirous of purely technical details are referred to the first part, which will shortly be published in the *Journal of Radiology*.

It might be well at this point to state our position as regards mastoid radiography in general. As brought out in the first part, we do not consider this method of examination as a means by which the otologist can shift the responsibility of making diagnoses or deciding for or against operation, and we believe that the radiologist who attempts to assume these responsibilities is in a fair way to discredit all mastoid radiography. We do know, however, that this is the only possible means we have of informing ourselves as to the anatomic structure of any given mastoid, unless we resort to mere guesswork or destructive exploration. As to the proof of this

statement, the hundred temporal bones so far examined were carefully studied externally and an estimate made as to internal structure before their subjection to X-rays. With all the advantage of no obtruding soft tissues coupled with exact knowledge as to the location of the sinus and the intracranial configuration, we were absolutely wrong in about 50 per cent of all cases. Certain types of infection in the living, to be sure, are coupled with certain anatomic peculiarities; but as to determining or even estimating the internal structure of a mastoid from its mere configuration, the above investigation speaks for itself.

Before proceeding with the main discussion, we wish to pay tribute to the kindness and courtesy of Dr. Randall, whose hearty cooperation has made this work possible.

Although it is well known that the structure of one mastoid may be totally unlike that of another, yet this fact receives little or no consideration in everyday practice. In the clinical study of a middle ear infection with possible mastoid involvement it is usually assumed, perhaps unconsciously, that the mastoid contains a certain amount of pneumatic cellular structure. That there is one chance in five that this is not the case was brought out by Cheatle,^{4 5} some fifteen years ago, when he demonstrated that 20 per cent of all adult mastoids contain practically no pneumatic cells, the "mastoid mass" remaining diploetic or dense as in infancy. These figures were recently confirmed by Turner⁶ and Porter, so far as European skulls are concerned. They also made the interesting observations that the percentage of infantile mastoids was greater in dolicocephalic skulls than in those of the brachycephalic type and that infantile mastoids were decidedly less prevalent in primitive races than in those on a higher plane of civilization. As the cell content of the individual mastoid ranges from none at all up to the most widespread distribution, these variations in structure apparently signify different degrees of development, with the infantile types at one extreme and the completely pneumatic at the other, while the numerous varieties of mixed mastoids represent the intervening stages.

It seems self evident that the extent of an acute mastoid infection together with its clinical course, in fact, its entire

pathology, must depend in large measure upon the amount and the distribution of the pneumatic cells. While there is some dispute as to how much reliance should be placed in X-ray findings as regards pathology, this is our sole means of information both as to the amount and the distribution of the pneumatic cells, short of mere guesswork or destructive exploration; though, judging by the X-ray reports as quoted in the current literature, this part of the examination is too often neglected. These reports deal with the question of haziness, cloudiness or the breaking down of cell walls and the like, but a report that gives the reader any definite idea as to the anatomic structure of the mastoid in question is decidedly rare. Just why important anatomic details that can be easily determined should receive so little attention is hard to fathom, but is probably due to inadequate and unstandardized methods for describing the same. As the usual classification of mastoids into three types, diploetic, pneumatic and mixed, is altogether too general for either description or comparison, a more precise classification would seem highly desirable. Several such have been proposed, one of which was devised by ourselves.¹ The latter is not purely arbitrary, but is based on the development of the mastoid, the various types described representing different degrees of development. It would seem as though such a classification ought to prove useful in describing and comparing the various types of mastoids encountered in everyday practice. Nevertheless, this classification, though perhaps possessing some merit, has proven, like all others, to be utterly useless for the purpose intended. This is due to the fact that the various types of structure are not so sharply differentiated as an abstract description would seem to indicate, but as actually encountered one type shades into another so gradually that clear cut divisions are impossible. Therefore, the only other alternative is to devise some practical method of accurately portraying and describing the anatomy of each individual mastoid in terms that are intelligible to the average reader.

The recent work of Wittmaack,² "The Normal and the Pathological Pneumatization of the Temporal Bone," supplies a terminology that is both comprehensive and easily understood, while the occipital X-ray projection, which discloses

all pneumatic cell structure, renders practically every mastoid amenable to concise and accurate anatomic description. Wittmaack has demonstrated that by the end of the first year the pneumatic structure of the mastoid consists of a large antrum with comparatively smooth walls. Under normal conditions pneumatic cells then began to develop in two directions: concentrically into the lumen of the antrum and excentrically toward the periphery. In concentric pneumatization, bony trabeculae sprout from the walls of the antrum underneath the mucous membrane. By their fusion one with another these trabeculae form first one layer of the fine pneumatic cells on the walls of the antrum and then others till the antral lumen becomes much narrowed. In excentric or peripheral pneumatization the original marrow spaces or diploe are replaced by pneumatic cells, all of which is described with the utmost histologic detail. As pneumatization progresses toward the periphery the cells become larger and larger, finally ending in a series of broad cul de sacs. These large peripheral cells are also partially subdivided by bony trabeculae that sprout up as in the antrum. This is a fact that should always be borne in mind when attempting to diagnose a pathologic breaking down of cell walls, for this appearance may be due to a normal but incomplete subdivision of the larger cells. In fact, such areas showing an apparent breaking down of cell walls are fairly common in the peripheral cells of absolutely normal mastoids. (Fig. 1, 2.) Radiograms of bones are used to illustrate this point because they can be reproduced much better than those from the living. However, the same appearance is frequently seen in the latter. If there be a certain amount of haziness in such cases, either from pathology or poor technic, there is quite likely to be some discrepancy between the X-ray reports and the operative findings, which probably accounts for some of the belief that mastoid radiograms are quite apt to be misleading.

According to Wittmaack, a mastoid that is not completely pneumatized by the end of the third year or, at the very latest, by the end of the fifth, never becomes so later on. Furthermore, in a completely pneumatic mastoid, the process of pneumatization never comes to a complete halt but continues on into old age, though at a decidedly slower pace.

In arrested development the disturbing factors—a detailed description of which is beyond the scope of this paper—have played their part by the end of the first year, and the earlier in life they come into play the more profound will be their effect on the process of pneumatization. In other words, restricted pneumatization is not due to infections that occur in later life. The following case has considerable bearing on this point: Figure 3 is a radiogram of Case 10 from our report of several years ago.¹ This was taken three and a half years after an acute mastoid infection that recovered without operation, though with some residual deafness and subjective symptoms. There is considerable density posteriorly, in the midst of which are scattered scraps of pneumatic cells, evidently sclerosis due to infection in adult life, as is seen when compared with the normal mastoid. (Figure 4.) Figure 5 is a radiogram of the same mastoid taken in November, 1922, or eight and one-half years after the original infection, and five years after the plate shown in Figure 3. While it may not be so evident in the reproductions, yet a comparison of the original radiograms shows a decided increase in the pneumatic cellular structure; whereas, according to our old conceptions, a decrease or at least a standstill is to be expected.

Wittmaack describes the completely pneumatic mastoid as follows: "In accordance with the somewhat different procedure in development, the cell net surrounding the antrum is relatively fine meshed, while the meshes gradually widen out toward the periphery and finally end in a fringe of large bladderlike cells. The partitions of this network consist of comparatively thin bone of about the same thickness." This conception of pneumatic structure as a cell net constitutes a reliable basis for describing the anatomy of all mastoids, irrespective of age or type of structure, providing all of the cell net be projected on to the X-ray plate and none of it be hidden by the petrous pyramid. In plates taken in the usual projection with the two meati superimposed, the pyramid extends backward and obscures the cell net in the vicinity of the antrum. (Fig. 6a.) While this may be of no great moment in large pneumatic mastoids, it is of the utmost importance in the examination of young children or those cases in which the extent of the cell net is decidedly limited. In the occipital

projection the pyramid is thrown forward and overlies the middle ear and the zygoma (Fig. 6b). As this obscures the zygomatic cells, both projections are necessary for a complete examination.

The foregoing description of the normal pneumatic cell net coupled with the occipital projection elucidates a point that has long defied our attempts at explanation, namely, the apparent presence of diploe about the antrum of otherwise completely pneumatic mastoids. Other observers have likewise been puzzled by this appearance, which has been described as a "dumbbell constriction in and about the antrum." Bearing in mind Wittmaack's description, it will be seen on comparing Figures 6a and 6b that this diploetic appearance is due to the fine meshed cell net, that normally surrounds the antrum, showing through the superimposed pyramid. These fine meshes of the normal cell net are apt to be mistaken for diploe, even in sectioned bones, as shown by Figure 6c, which is the same bone as in Figures 6a and 6b. On comparing these three figures, it will be seen that the occipital projection roughly corresponds to a vertical cross section of the mastoid in the sagittal planes.

The mastoid with no pneumatic cells presents the greatest contrast to the completely pneumatic type, as shown by Figure 7a. This might be termed either a diploetic, an infantile, an acellular or a sclerosed mastoid or one with total arrest of pneumatization; but if the radiologist simply state that the entire mastoid is diploetic with no visible evidence of pneumatic cells, he will give us a clearer concept of its actual structure than if he merely give it a name. From this information the otologist will be able to visualize the anatomic relations fully as well if not better than by exploratory operation. He will know that the pneumatic structure of such a mastoid consists of an antrum deeply placed in dense bone; that there may be only a very thin plate of bone between the antrum and either the middle or the posterior fossa or the sinus or all three combined; that, as even the most virulent infection in an antrum so situated is incapable of producing but few if any of the typical signs and symptoms of mastoiditis, though all the more likely to give rise to intracranial

complications, an operation will be urgently indicated on far less clinical evidence than almost any other type of mastoid. In other words, he should not allow a patient to die of meningitis or some other intracranial complication while waiting for typical signs and symptoms of mastoiditis to make their appearance. Even though delay be unattended by disaster, it should always be borne in mind that with this type of mastoid a middle ear discharge is quite likely to become chronic. Hence, in view of the constant annoyance, the loss of hearing and the ever present menace to life thereby entailed, a simple mastoidectomy is often indicated for the prevention of this complication, even though there be no other sign or symptom, save otorrhea, to justify its performance.

That the occipital projection (Fig. 7a) is a reliable guide as to the absence of cells is demonstrated by sectioning the bone (Fig. 7b). From the standpoint of development, it makes little or no practical difference whether the mastoid be dense or diploetic. In calling attention to the two infantile types, the dense and the diploetic, Cheatle^{4 5} emphasizes the fact that in the latter a layer of compact bone always separates the antrum from diploe. He offers no explanation as to the occurrence of these types beyond suggesting an increased density of bone as a determining factor. From Wittmaack's description² of pneumatization it becomes evident that the layer of compact bone separating the antrum from diploetic structure is due to a conversion of the narrow spaces or diploe adjacent to the antrum into compact bone; while, in the case of the dense mastoid, this process is so exaggerated that the entire mastoid becomes sclerosed. In other words, these two types, the diploetic and the dense, are sclerosed mastoids due to total arrest of pneumatization, differing only in the degree of sclerosis. It seems evident, however, that osteomyelitis would be more probable in a diploetic than in a dense mastoid, especially if there be a small amount of pneumatic structure by which an antrum infection could be carried deeper into the mastoid. Wittmaack has further demonstrated that the bone itself plays but a secondary role in the process of pneumatization. If the bone be dense the cells may be smaller and the cell net not so extensive, but in this case, if other conditions be normal, the entire mastoid will be small yet perfectly pneumatized. The

occurrence of perilabyrinthine cells is added evidence that the density of bone per se is no bar to pneumatization, as the densest bone in the body is to be found in this region.

A very small fine meshed cell net is depicted in Figure 8a. This radiogram demonstrates the value of projecting the pyramid forward so as not to obscure the region of the antrum, as may be seen on comparing this radiogram with that shown in Figure 8b, taken in the usual angle with the two meati superimposed. That the occipital projection discloses practically all of the pneumatic cells that are present is again demonstrated by sectioning the bone (Figure 8c). This is of the greatest importance wherever the cell net is much restricted, as is usually the case in very young children. The superiority of the occipital projection at this age becomes apparent on comparing the mastoid radiograms of a twenty-two months old child (Figure 9a, 9b), taken with the usual and the occipital angles respectively. Whether or not this be brought out in the reproductions, a study of the original films discloses in the occipital projection a small but well marked cell net in the vicinity of the antrum, practically all of which is hidden by the pyramid when the two meati are superimposed. These two mastoids, the one in the dry bone and that in the child, can be accurately described as follows: X-ray examination discloses a much restricted cell net limited to the vicinity of the antrum, the remainder of the mastoid being entirely diploetic.

Infection in a mastoid of this type at this age or thereabouts will usually evoke the typical signs and symptoms of mastoiditis, though our experience has taught us not to rely on this too implicitly. In the case of the adult, however, we should always remember that even this grade of arrested development may be coupled with sclerosis sufficient to mask the typical signs and symptoms of mastoiditis almost as effectually as in the acellular types. Hence the slightly pneumatized mastoid is, potentially, the most dangerous of all; for the small amount of pneumatic structure predisposes to a deeper and, at the same time, to an insidious extension of virulent infection.

A more advanced stage of development is shown in Figure 10. The cell net is typical in that its meshes are small in the antral region and gradually widen out toward the periphery.

It is limited, however, to the upper two-thirds of the mastoid, the tip being entirely diploetic. This illustrates another important point brought out by Wittmaack in the course of his investigations, namely, that while the upper part of the mastoid is pneumatized by cells that push out from the antrum, the tip is pneumatized from the hypotympanum. The aditus serves as the outlet to the middle ear for the upper group of cells, while the sinus tympani, located in the posterior pole of the hypotympanum, performs this function for those in the tip of the mastoid. In the normal course of development one cell system so blends with the other that the difference in outlet becomes unimportant.

Complete pneumatization of the upper two-thirds of the mastoid with a total arrest at the tip is fairly common. The reverse of this condition, while possible, seems to be extremely rare, and we have found no reference in Wittmaack to any such type of structure. Be that as it may, within the past year we encountered at operation a case of this type in which, unfortunately, the indications for operation were too evident and too urgent to permit of an X-ray examination. The upper half of the mastoid was entirely dense, while the entire tip and the bone for an inch posteriorly contained large pneumatic cells filled with pus. A chain of small deeply placed cells extended upward along the posterior canal wall and ended abruptly at the approximate level of the hypotympanum, though a due respect for the facial nerve prevented an exact determination of this point. It might be interesting to note that this patient, a child of twelve, had been in extreme pain for two weeks with no aural discharge at any time or, for that matter, no paracentesis. When examined previous to operation the drum, though congested, showed no bulging. Though mastoids that are dense or diploetic in the antral region and pneumatic below seem to be most unusual, those whose antral cell net is much restricted while the tip is completely pneumatic are of comparatively frequent occurrence. A good example of this condition is shown in Figures 11a, 11b and 11c.

In a previous communication¹ we described a type of mastoid, long known to otologists of the Massachusetts Charitable Eye and Ear Infirmary as the "double decker." The cells of

a "double decked" mastoid are arranged in two tiers, an inner and an outer, which are separated by a layer of bone closely resembling the inner table. We attempted to explain this type as a mixed mastoid with diploe in one plane and pneumatic structure in the other, but in the light of Wittmaack's* observation that the mastoid is pneumatized not by one cell system alone but by two which, at first, are separate and distinct, it seems probable that the "double decked" arrangement is due to the antral cell system failing to blend with that from the hypotympanum. Occasionally the squama, which forms the outer antral wall and approximately the upper third of the mastoid cortex, extends downward over the petrosa nearly to the tip of the mastoid. It seems possible that this overdevelopment of the squama may be coupled with an increased resistance along the plane of the squamomastoid suture sufficient to explain a lack of fusion of the two cell systems. At all events, this type of mastoid generally has a few large cells at the tip, and at operation the "double decked" condition is usually unsuspected until these tip cells are uncapped. As the clinical significance and the X-ray examination of this type of structure have been fully discussed in the previous communication,¹ to deal with them here would be mere repetition. It seems well to state, however, that the radiologist who does not make stereoscopic study a part of his routine examination is not likely to forewarn the otologist as to this type of structure.

Again consulting Figure 10, it will be seen that in addition to the diploetic tip the cell net is somewhat restricted posteriorly, the cells in contact with diploe having thickened walls. According to Wittmaack, this latter condition is characteristic of arrested pneumatization due to hyperplasia of the lining mucosa. This increased density of bone at the periphery of the cell net, though probably serving as a barrier against the spread of infection by contact, has other accompaniments that render the intracranium more susceptible to blood stream infection. As an explanation of these points involves the entire question of mastoid development, it seems proper to

*Wittmaack does not mention the "double decked" type of mastoid.

digress for the moment in order to mention the salient histologic features of this process.

The fetal middle ear cavities—tympanum, recessus and antrum—are first blocked out by bony—that is to say, cartilaginous walls, and are filled with a gelatinous embryonic tissue known as mesenchyme. A lumen begins to develop between the fifth and sixth month of fetal life by an evagination of epithelium from the eustachian tube. As the lumen expands the embryonic tissue recedes so that the mucous membrane that lines these cavities is, at first, normally hyperplastic and consists of epithelium originating from the eustachian tube and subepithelial tissue derived from mesenchyme. During this time capillaries, channeling in from the jugular bulb, from the dura of both fossæ and from the knee of the lateral sinus, come into intimate contact with the submucosa of the developing pneumatic cavities and their respective cell systems. Under normal conditions, this hyperplastic mucosa regresses to a mucoperiosteal membrane of such thinness that individual layers can no longer be differentiated, the vascular communications with the dura, the sinus and the jugular bulb being thereby obliterated. In the tympanum, the recessus and the antrum this regression is either completed by the end of the first year or probably not at all; if the mucosa remain hyperplastic its vascular connections also persist. The completion of the middle ear cavities marks the end of the first period of development; the second, which is characterized by the formation of the pneumatic cells, closely follows.

Both the hypo- and epitympanic as well as the antral cells are formed in two ways: First, by the fusion of bony trabeculae that sprout from the walls of the respective cavities, followed by evagination of the lining mucosa into the spaces thus formed; second, by progressive transformation of the adjoining marrow spaces into pneumatic cells. Before these spaces actually become pneumatic they undergo preliminary transformation. As the mucosa of the middle ear cavities has previously regressed to a thin mucoperiosteal membrane the mucosa adjacent to a marrow space, about to be pneumatized, becomes vascular and somewhat thicker. A cone of this thickened subepithelial tissue then forces its way into the marrow

space and displaces the marrow content. The continued growth of this cone causes an absorption of bone, with the result that several spaces are thus thrown into one. The perivascular tissue of capillaries from the dura of both fossæ, the lateral sinus and the jugular bulb, respectively, also take part in this preliminary process. Hence, when the former marrow space becomes pneumatic by evagination of epithelium from the adjoining pneumatic cavity the mucous membrane that lines the newly formed cell is at first normally hyperplastic and has the same vascular connections as the respective middle ear cavity from which it originated. These are, likewise, severed when the mucosa regresses to a thin mucoperiosteal membrane; but if regression does not occur and the mucosa remains hyperplastic these vascular communications with the dura, the sinus or the jugular bulb also persist. Moreover, instead of an absorption of bone, as at first, a deposition of bone then ensues, causing the cell walls to become much thickened. If, during the first year, when the mucosa of the middle ear cavities is normally hyperplastic, some disturbing influence prevents its complete regression or renders it even more hyperplastic, the mucosa may be so thick that evagination of epithelium into the spaces prepared for the same becomes impossible. As this hyperplasia is limited to the superficial layers of the mucous membrane it does not interfere with preliminary transformation of the marrow spaces but, on the contrary, actually favors this process, so instead of these spaces being further transformed into pneumatic cells they are eventually converted into dense bone. In the case of the diploetic mastoid this explains the layer of compact bone that separates the antrum from diploe. So far as their development is concerned there is no essential difference between this and the completely dense mastoid, except that in the latter type the sclerosing process has been more active.

At first glance it would seem as though a deposition of bone sufficient to obliterate the marrow spaces would also obliterate the vascular connections of a hyperplastic mucosa, but such is not likely to be the case, for when bone opposes a blood vessel the bone, not the vessel, succumbs. An aneurism, for example, will invariably erode any bone in the body that tends to obstruct its growth. Therefore, whenever the radiogram dis-

closes an infantile type of structure or a restricted cell net whose peripheral cells show thickened walls the possible vascular communications with adjacent intracranial structures should be kept in mind. Dry temporal bones with restricted pneumatic development frequently show numerous pin point openings at the knee of the sinus. Whether or not these openings represent direct vascular communications between the mastoid and the sinus in the living is of course open to question, though they are certainly suggestive. This point could probably be determined by injecting fresh material or possibly wet specimens.

As mentioned before, the tip of the mastoid is pneumatized not from the antrum but from the hypotympanum. In the preliminary transformation of the marrow spaces in this region the process begins at the sinus tympani and, proceeding backward and downward, takes in the marrow spaces surrounding the facial nerve. This is a point well worth remembering when extirpating the deeper cells of the posterior canal wall.

While certain disturbing influences* may affect the superficial layers of the mucosa with consequent persistence or even an exaggeration of the original hyperplastic condition, other causes may give rise to fibrosis of the deeper layers without superficial effects. In this case the submucosa will be inca-

*Wittmaack believes that hyperplasia of the mucosa is caused by the entrance into the tympanum of amniotic fluid, meconium, etc., during fetal life, or more probably at birth; or the entrance of vomitus, etc., during the first year of life; that this material sets up a foreign body irritation which is coupled with a low grade infection that is insufficient to produce symptoms such as perforation and discharge. Wittmaack believes that this accounts for the great frequency with which collections of pus are found at autopsy in the middle ears of young children. Solowzow found this condition in over 90 per cent of all children coming to autopsy. It has been designated otitis media concomitans, otitis media neonatorum and otitis media of the suckling. Fibrosis, on the other hand, he believes to be due to acute middle ear infection during the first year of life.

The recent investigations of Dean and his workers are most interesting. They have found collections of pus and a thickened mucosa in the sinuses and the middle ears of rats fed on a diet deficient in fat soluble vitamin A. This work is suggestive, at least, as regards otitis media neonatorum and the etiology of a hyperplastic mucosa.

pable of carrying out a preliminary transformation of the marrow spaces, while the epithelium may sink into them directly. In this event the pneumatic cells thus formed will be small and limited in extent (Fig. 12), though not to the same degree as in cases with intense hyperplasia. Such pneumatic cells will have neither the thickened walls nor the vascular connections that are found in arrested pneumatization with a hyperplastic mucosa. There are probably innumerable cases of arrested development in which both factors come into play.

This in barest outline is the process of normal and abnormal pneumatization, as described by Wittmaack, and is thrust into this particular paper because of its extreme importance in the interpretation of mastoid anatomy.

Another type of structure that requires mention is the over-pneumatized mastoid. (Fig. 13a.) Wittmaack believes this is due to a hyperplasia of the mucous membrane too slight to prevent or even hinder evagination of the epithelium and yet sufficient to so stimulate preliminary transformation of the marrow spaces that not only the mastoid proper but the bone adjacent becomes widely pneumatized. The usual projection is more useful than the occipital for the study of this type of mastoid, as will be seen on comparing Figures 13a and 13b. The upper portion of the mastoid shown in Figures 14a and 14b is extensively pneumatized, while the tip is entirely dense. On the basis of Wittmaack's theories, this indicates that during the course of development there was a slight hyperplasia of the mucous membrane of the antrum sufficient to induce an overdevelopment of the antral cell system, while the mucosa of the hypotympanum was so intensely hyperplastic as to prevent pneumatization of the tip.

If the mucosa be somewhat more hyperplastic than was, presumably, the case in Figures 13a and 13b and the upper portion of Figures 14a and 14b, the cell net may be normal in extent, but with partitions between cells much broader and more diploetic than under normal conditions. This kind of structure is shown at the periphery of the cell net in Figures 15a and 15b. Though a mastoid of this type appear completely pneumatic, it may in reality be a mixed mastoid with

the pneumatic structure intermingled with diploe, in contradistinction to the other variety in which the two are segregated. (Figs, 8, 9, 10.)

Under these conditions it would seem quite possible for a mastoid infection to set up a low grade osteomyelitis in the framework of the cell net that might remain dormant for some time after typical signs and symptoms had disappeared, a condition that might account for some of the cases of so-called "latent mastoiditis." As it is obvious that no hard and fast line can be drawn between this and the completely pneumatic mastoid, the added information to be gained from stereo plates is usually necessary in order to distinguish between these two types.

The structure of the pneumatic mastoid permits of rapid and widespread infection, which often dies down about as quickly as it flares up. This behavior is usually attributed to the large size of the cells, which is presumed to insure good drainage—a most inadequate explanation at best—which in the light of present knowledge becomes absolutely untenable. For how can their mere size favor the drainage of the mastoid cells, inasmuch as they must all drain through the fine meshed cell net that surrounds the antrum?

As mentioned in our communication¹ of several years ago, changes in aeration that result from a middle ear and mastoid infection probably account both for the abrupt onset of stormy and threatening symptoms and for the rapid subsidence of the same, so often observed in the case of pneumatic mastoids. As an otitis media first renders the eustachian tube impermeable, absorption of air from the middle ear cavities and the pneumatic cells naturally follows, while the resulting venous congestion of the lining mucosa is the best possible preparation for a rapid spread of infection. It seems probable, however, that incision of the drum membrane or even spontaneous perforation not only promotes drainage but also tends to re-aerify the middle ear and the mastoid cells, thereby restoring the air pressure within these cavities, approximately, to that of the surrounding atmosphere. This, while reducing congestion, probably tends to expel secretions from the mastoid

cells, with the result that patients not infrequently recover before the final arrangements can be made for mastoidectomy.

As the mastoid begins to re-aerify, the X-ray discloses isolated cells in the vicinity of the antrum that are clear as contrasted with the general haziness of the rest of the mastoid. When this appearance is found while the clinical symptoms are at their very height, as is sometimes the case, marked improvement usually follows within the next twenty-four hours. Such cases, however, do not always go on to complete recovery. Those that do not probably contain diploe, either in the cell walls or at the periphery of the cell net, in which an osteomyelitic condition has become so firmly established that re-aerification of the pneumatic cells does not suffice to restore the mastoid to normal; while, in the completely pneumatic mastoid, if the infection has not actually destroyed the cell walls, reestablishment of the normal air content is probably all that is required to bring about healing. In addition to purely mechanical factors the beneficial effect of oxygen on the suppurative process is to be considered.

SUMMARY.

We believe the foregoing demonstrates that variations in anatomy are dependent on the degree of pneumatic development. A study of our complete series of X-ray films is even more impressive in this regard, for they show about every possible gradation, all the way from the completely acellular mastoid to the most widespread pneumatization, while the anatomic variations described and illustrated in this paper are merely the outstanding types of structure. In view of the fact that the structure of one mastoid is likely to vary so widely from that of another, it seems self-evident that in every serious middle ear infection the anatomy of the mastoid in question should be considered along with the clinical evidence in the case. Our textbook descriptions of the pathology and the symptomatology of mastoiditis together with the indications for operation are all based on the typical mastoid. As there is apparently no such thing as a "typical" mastoid, it would seem as though these descriptions should be revised so

as to conform more closely to the main types of mastoid structure as they actually occur.

We believe that Wittmaack's description, both of pneumatization and of types of structure, provides a most satisfactory basis for the interpretation of mastoid anatomy. However, we do not wish to give the impression that we accept his theories and conclusions unreservedly, for one thoroughly familiar with his work will see that this paper is based chiefly on his observations, with but incidental reference to his theories and conclusions. We are fully aware of the objections that can be raised against the latter, but to reject the entire work for this reason alone seems to us most illogical, for in passing judgment on its worth three factors should be considered. First and foremost, do these observations and investigations give us the actual facts as regards complete and incomplete pneumatic development or, as Wittmaack expresses it, regarding normal and pathologic pneumatization? As to this, a review of the literature and consultations with several eminent embryologists have disclosed, so far as we have been able to determine, no investigations whatever in which the histologic processes of pneumatization have been observed and described. We therefore believe that Wittmaack's observations in this regard are not to be rejected unless disproven by subsequent investigations of a similar nature. If we but familiarize ourselves with Wittmaack's work we are then fully able to form our own conclusions as to its practical applications, and whether or not our conclusions agree with those of Wittmaack seems of little consequence. Furthermore, whether we accept his explanation as to the causes of incomplete pneumatization, or whether we can offer a more logical explanation of our own, or whether we hold this question in abeyance seems to us of relatively minor importance.

Without going into purely technical details, it seems well to conclude with a few general remarks as to X-ray examination. If this is to be of any real use to the otologist, it is obvious that it must be adequate as well as technically perfect. We ourselves do not believe that the structure of any mastoid, with the possible exception of infants, can be accurately determined by means of one flat plate, no matter how projected

or how perfect the technic. After a thorough trial of the projections devised by others as well as those devised by ourselves, we believe that an adequate examination requires three plates for each mastoid: a stereoscopic set in the usual angle with the two meati superimposed and a flat plate in the occipital projection. The former shows both the structure and general relations of the mastoid in three dimensions, while the latter discloses whatever cell net be present in the mastoid proper, much the same as would a vertical cross section in the sagittal plane. If conditions be such that but one plate can be taken, we have found the occipital projection to be the most useful, especially where repeated examinations are indicated. If double intensifying screens and films be used the radiation otherwise required is so reduced that repeated examinations can be made without danger.

While in nowise minimizing the pathologic conditions that may be disclosed by X-ray examination, we are firmly convinced that focusing the attention solely on this feature has been a potent means of discrediting mastoid radiography in the eyes of many otologists; for, as a matter of fact, the well trained otologist does not need to have the radiologist make his diagnoses or tell him when to operate. We do believe, however, that regardless of age or type of structure, an accurate foreknowledge of mastoid anatomy can be obtained by means of the X-ray and by this means only. Given this information, it is possible to determine the grade of development and to form some conception of the histologic structure of the individual mastoid. These questions then cease to be purely academic, but on the contrary, if brought to bear on the everyday problems of mastoid disease, it would seem as though diagnosis, prognosis and the establishment of indications for operation would be placed on a more scientific, less empiric basis than is otherwise possible.

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FIGURE 1.

Large cell in tip. Incomplete trabeculation, simulating broken-down and frilled-out walls.

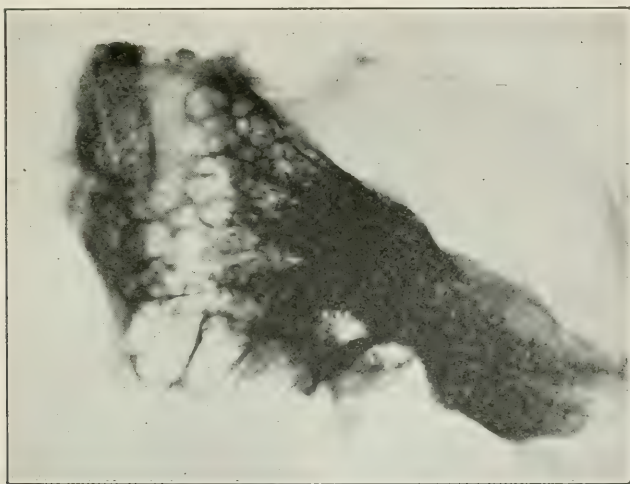


FIGURE 2.

Same condition as Figure 1, somewhat more pronounced.

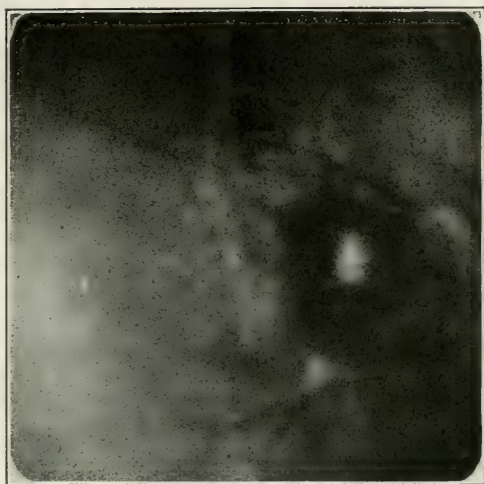


FIGURE 3.

Mrs. H. Examined in 1917, three and one-half years after an acute mastoid infection. Recovery without operation. Considerable density posteriorly, in midst of which are scattered scraps of pneumatic cells.

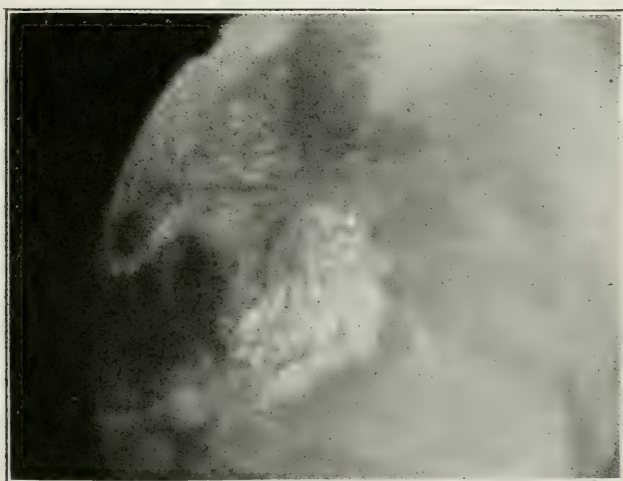


FIGURE 4.

Mrs. H. Left normal mastoid, showing completely pneumatic type.

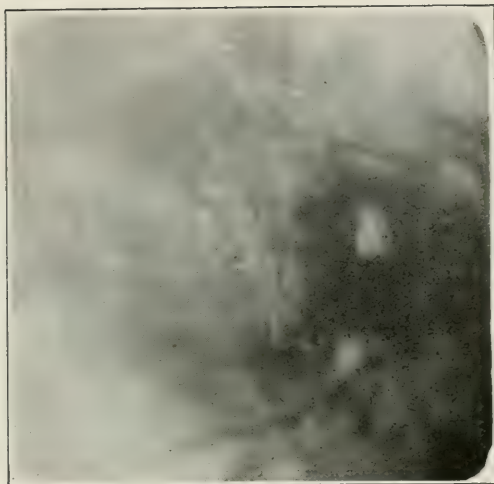


FIGURE 5.

Mrs. H. Right mastoid, examined in 1922, five years after Figure 3. Definite increase in cellular structure.



FIGURE 6-A.

Completely pneumatic mastoid, projected in usual manner with internal and external auditory meati superimposed. Note how petrous pyramid is projected backward, obscuring region of antrum. (Arrow.)

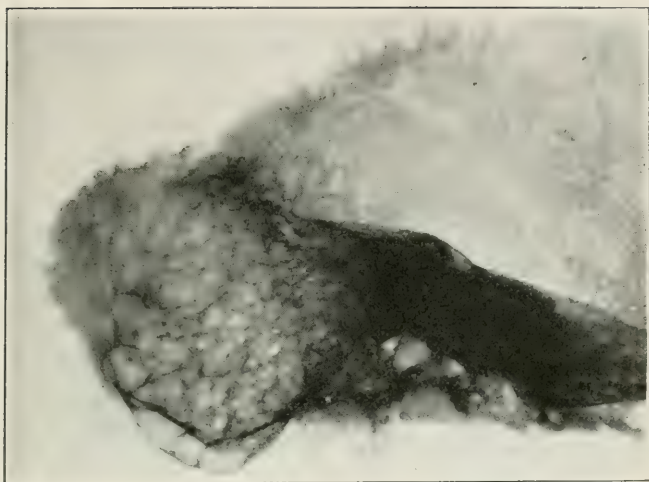


FIGURE 6-B.

Same mastoid, taken with occipital projection. Pyramid is projected forward over tympanic cavity and temporomandibular joint. Antral portion of mastoid unobscured. Note fine meshed cell net in this region.

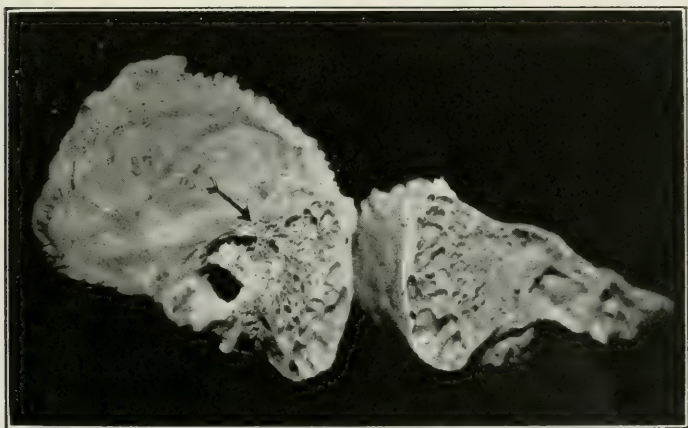


FIGURE 6-C.

Same bone in vertical cross section, sagittal plane. Note that occipital projection (Fig. 6-B) shows approximately same cell distribution as this sectioned bone. Arrow points to fine meshed cell net in outer antral wall, resembling diploe.

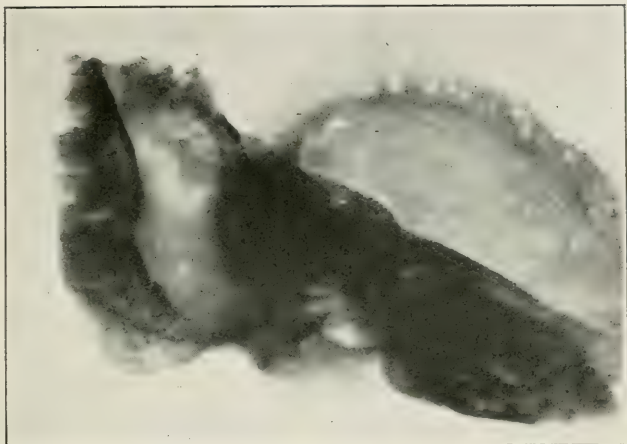


FIGURE 7-A.

Occipital projection showing complete absence of pneumatic cells. Lateral sinus very prominent, due to superficial and forward position.

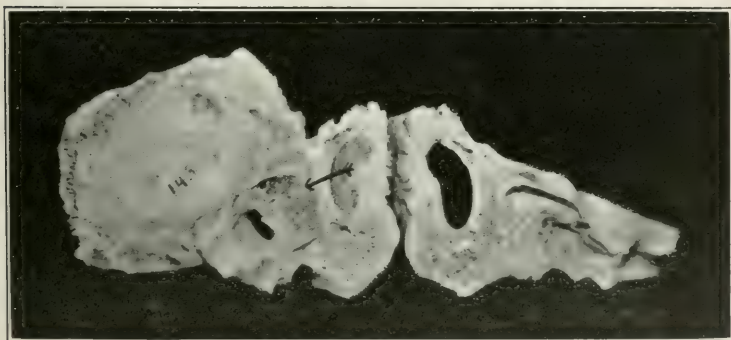


FIGURE 7-B.

Same bone sectioned. Arrow points to compact bone separating antrum from diploe. Several tiny depressions are seen in outer antral wall.



FIGURE 8-A.

Occipital projection, showing a fine meshed cell net (arrow) in immediate vicinity of antrum.

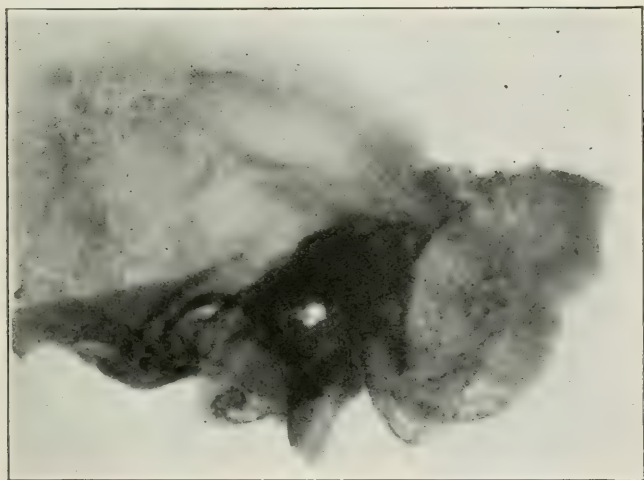


FIGURE 8-B.

Regular projection. Cell net almost entirely obscured by backward projection of petrous pyramid.

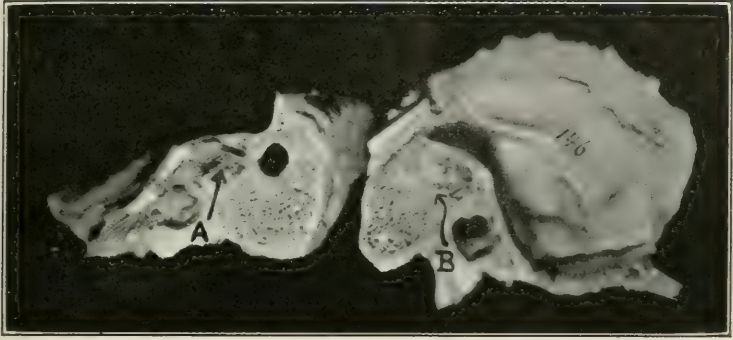


FIGURE 8-C.

Sectioned bone. A—Fine meshed cell net about antrum. B—Compact bone separating antrum from diploe.

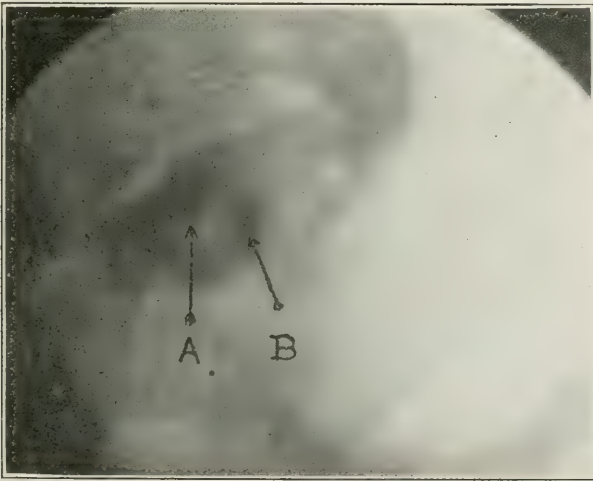


FIGURE 9-A.

Child 22 months old. Regular projection. A—Tympanic cavity. B—Pyramid obscuring antral region.

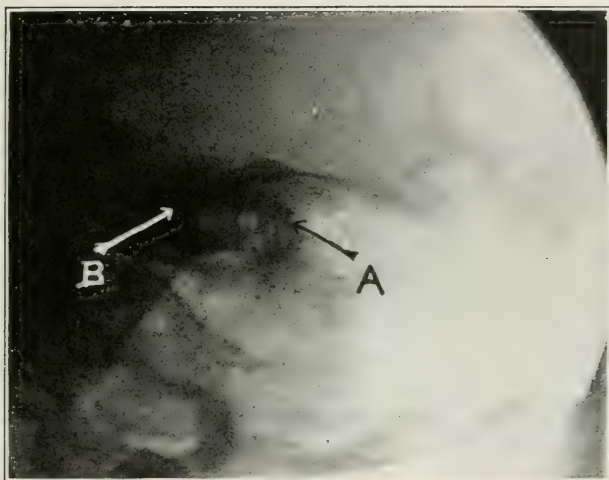


FIGURE 9-B.

Same child, occipital projection. A—Small but definite cell net seen about antrum (arrow). B—Pyramid projected forward.

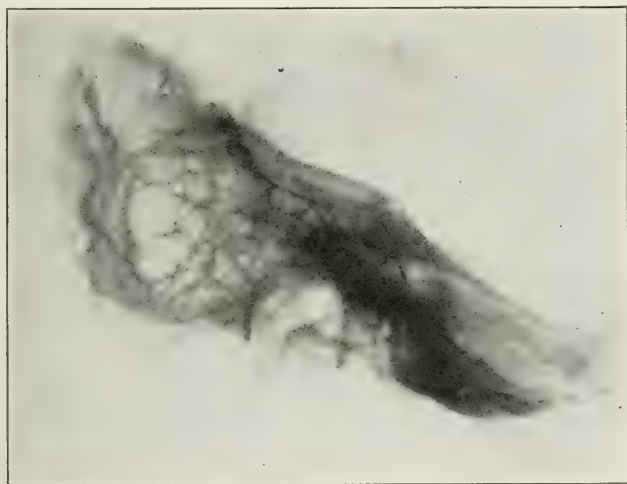


FIGURE 10.

Occipital projection. Upper two-thirds shows typical pneumatic cell net, with fine meshes in region of antrum widening out toward periphery. There is some limitation of cell net posteriorly, while tip is entirely diploetic. Note thickening of peripheral cell walls.

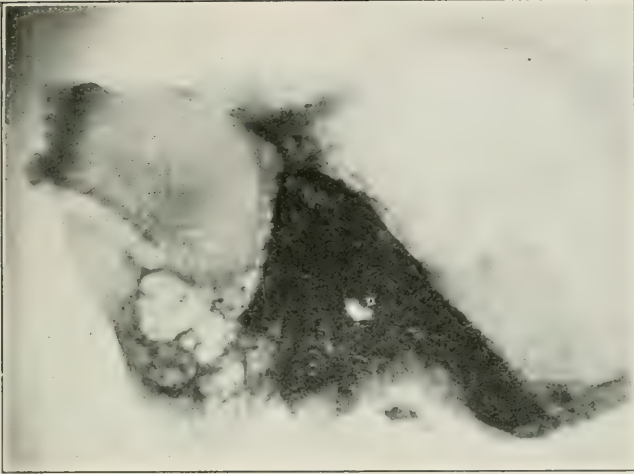


FIGURE 11-A.

Usual projection. Tip completely pneumatic. Considerable limitation of cell net in upper half.

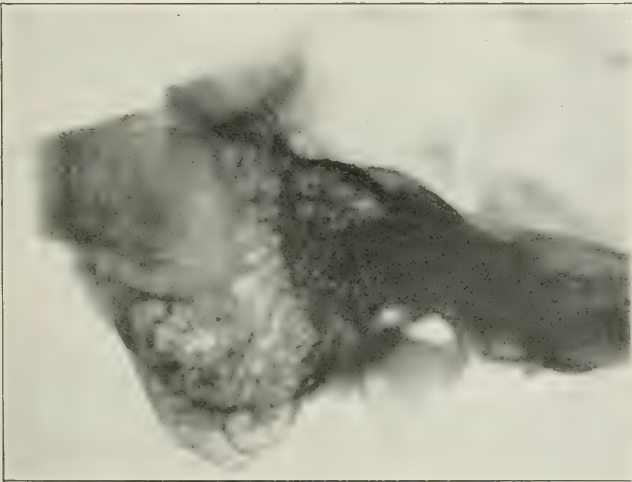


FIGURE 11-B.

Occipital projection. Partial development of upper half seen more clearly. This demonstrates superiority of occipital projection in depicting region of antrum.

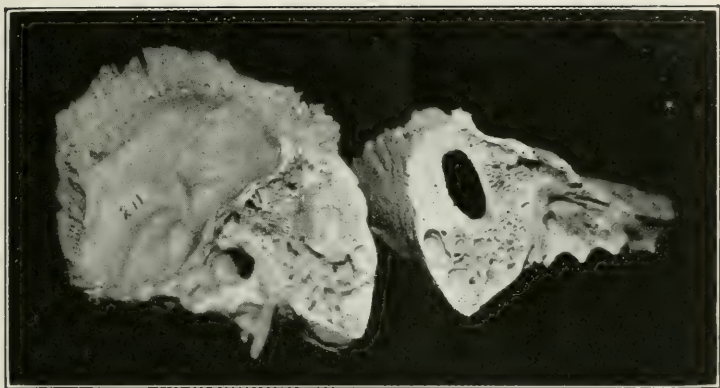


FIGURE 11-C.

Vertical cross section, sagittal plane. Again demonstrates that occipital projection brings out cell net practically as well as section of bone. (This bone was sectioned further, but no additional pneumatic cells were found in upper half.)



FIGURE 12.

Cell net very much limited in extent. All cells are small, without thickening of peripheral walls. Lateral sinus superficial and forward.

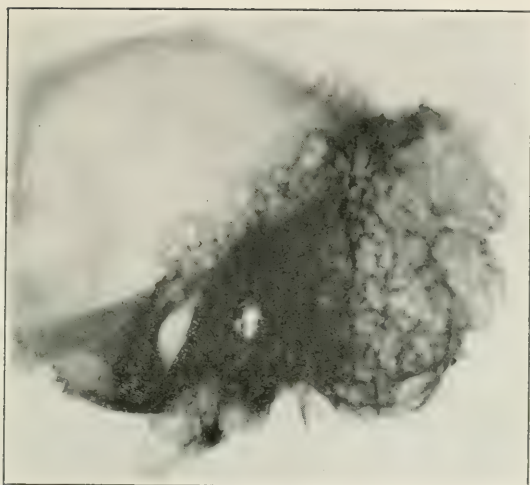


FIGURE 13-A.

Usual projection. Extensive pneumatization, cells reaching through zygoma to temporomandibular joint.

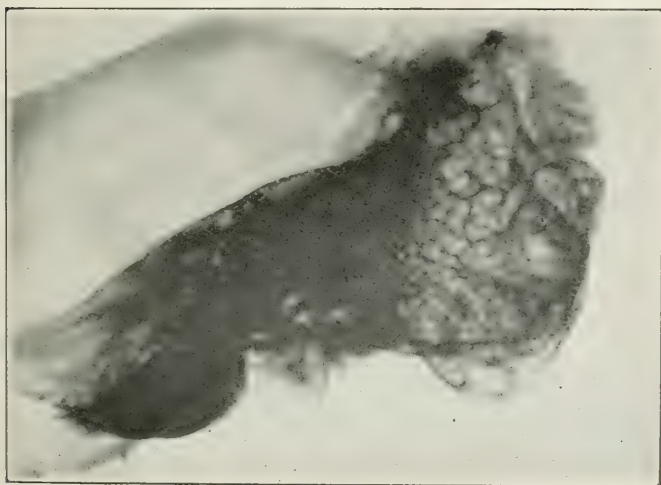


FIGURE 13-B.

Occipital projection. This view does not reveal cells in region of zygoma as well as usual projection.

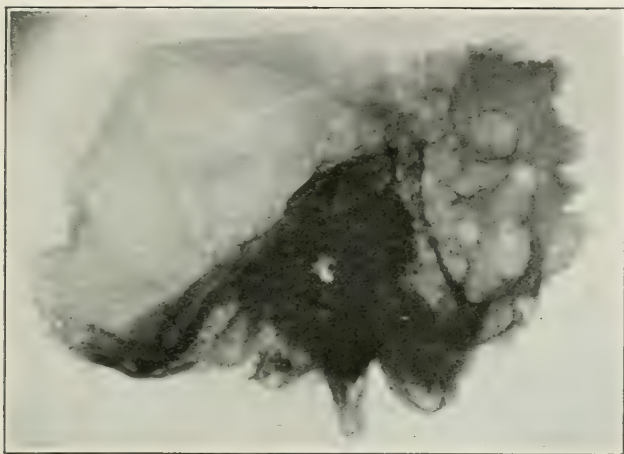


FIGURE 14-A.

Usual projection. Extensive pneumatization of upper portion, while tip is completely diploetic.

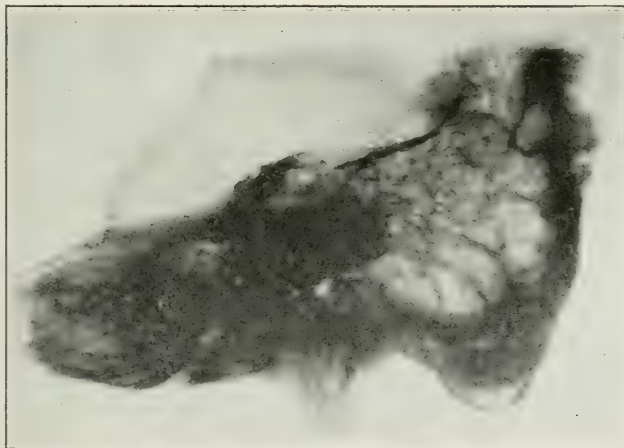


FIGURE 14-B.

Occipital projection. Shows sharp limitation of penumatic structure and thickened peripheral walls near tip.

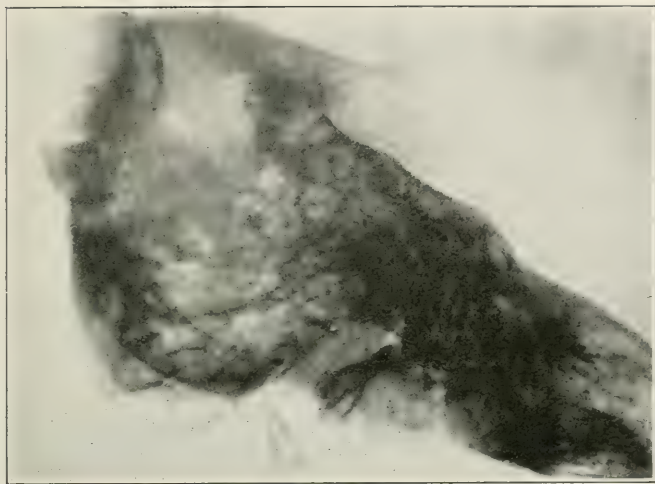


FIGURE 15-A.

Occipital projection. Cell net shows limitation posteriorly. All cells are thickened.

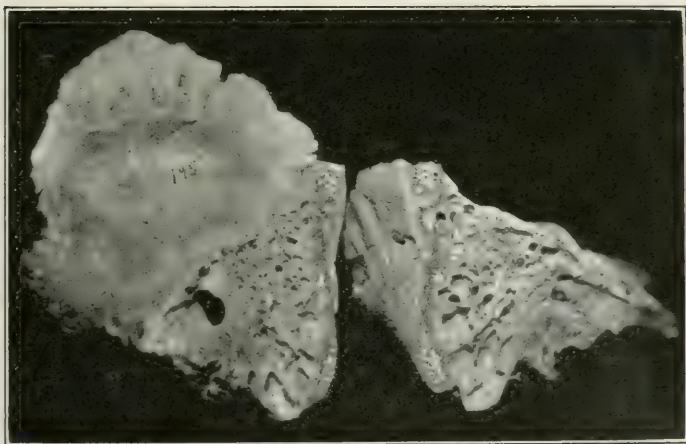


FIGURE 15-B.

Sectioned bone, showing same condition.

L.

THE INCIDENCE AND HISTOPATHOLOGY OF BONE AND CARTILAGE IN THE TONSIL.*

BY CARL VERNON WELLER, M. S., M. D.,

ASSOCIATE PROFESSOR OF PATHOLOGY, UNIVERSITY OF MICHIGAN,

ANN ARBOR.

To the clinician, the occurrence of larger or smaller nodules of bone and cartilage in the tonsillar capsule and stroma, palpable to the examining finger or mechanically interfering with operative procedures, has always been a matter of great interest. Similarly, the histopathologist, whose microscopic sections reveal an altogether unexpected incidence of such nodules, many of them so small that they could not be detected otherwise, finds that their occurrence and histogenesis throw light upon an important part of the pathology of the tonsillar region. Although Barnes² dismisses the finding of bone and cartilage in the tonsil with a word, the increasing bibliography upon this subject, and especially the persistent appearance in the same journals of successive articles dealing with it, bear witness to the interest which it arouses. The latter point may well be illustrated from the *ANNALS*, in which there have appeared the papers of Grosvenor⁹ (1908), Kelsey¹⁴ (1911), Grove¹⁹ (1916), Albert and Douglass¹ (1917) and Forman⁷ (1918). That such a sequence of reports seemed desirable and necessary has been due to the fact that no one of them has decisively established either the frequency of occurrence of such nodules of cartilage and bone or the relative parts played by chronic inflammation and inherent tissue potentiality in their genesis.

It seems unnecessary to give an extended review of the literature of bone and cartilage in and about the tonsil since the first reported cases of Orth²⁰ in 1893. A bibliography is appended and to its more important items reference will be made at appropriate points. However, there are three important aspects of this literature to which general reference must

*From the Department of Pathology, University of Michigan.

here be made, since they mark stages of progress in the growth of this subject. Many of the earlier papers report the occurrence of gross islands of bone and cartilage, such as interfered with the removal of the tonsil, and attention was paid to the continuous or discontinuous relationship of these to the styloid process. In another, and in part simultaneously appearing group of papers, we find case reports of a few instances, perhaps only one or two, in which bone or cartilage was discovered in the course of microscopic examination of the tonsil. In the erroneous belief, based upon a limited experience, that such a finding was highly unusual, a single observation was considered worth reporting. In the third place, from the standpoint of etiology, the earlier literature was highly controversial, and arguments for and against metaplasia associated with chronic inflammation as contrasted with developmental disturbances and growth of "embryonal rests" play an important part; while in more recent papers increasing attention is given to the embryologic history of the parent tissues and their inherent powers of proliferation and metaplasia.

Material and Method.—About 15,000 pairs of faucial tonsils have now been examined microscopically in the pathologic laboratory of the University of Michigan. These have been fixed routinely in formol, into which they are placed in the operative clinics. Upon receipt in the laboratory they have been bisected longitudinally in a plane at right angles to the oropharyngeal surface, and one-half of the tonsil has been embedded in paraffin for sectioning, regardless of its size. The sections have been cut from the surface produced by the bisection, thus giving the largest possible area of tonsil tissue for examination and one which presents at the same time surface mucosa, longitudinal and cross sections of the ramifications of many of the crypts, the septa between crypt systems and the capsular structures. Such an enormous material can not be examined in serial sections, but experience has shown that pathologic changes of importance will rarely be missed in a systematic field-by-field examination of a few sections oriented according to the method developed in this laboratory. Hemalum and eosin staining has been employed routinely, with special stains as indicated.

In a majority of the cases the original pathologic diagnoses

were made by Dr. A. S. Warthin, and upon this accumulated data I have been permitted to draw freely. For the statistical purposes of this article, however, the writer has made a systematic field-by-field reexamination of the sections from 1,000 consecutive pairs of tonsils with special attention to the points here considered. It so happened that in these 1,000 consecutive cases there were 76 in which the age was not known. In order to have age incidence data for 1,000 cases available, 76 additional consecutive cases of known age were included. Two series of cases are therefore considered in appropriate sections of this report: 1,000 consecutive cases and 1,000 consecutive cases of known age. As will be pointed out, there were slight differences due to chance distribution in the bone and cartilage incidence in the two series. In a very few cases one tonsil only was received, but this occurred so infrequently that it need not be considered in a mathematical analysis of the material.

The large number of cases here considered, far in excess of any other report upon this subject, renders the factor of chance distribution negligible; and the consecutive nature of the series chosen for statistical study nullifies any criticism based upon the fallacy of random sampling. Thus the two forms of error, most often met in medical statistics, are avoided. There are, however, serious sources of error, inherent in the method itself and of chief importance in their effect upon the percentage of incidence of bone and cartilage. These all operate in the same direction, depressing the ascertained incidence below the actual fact, so that all figures here presented represent a minimum value demonstrably lower to an unknown degree than the true incidence. For example, bone and cartilage may be left behind in the course of removal of the tonsil, or may tear out in the cutting of the section through their greater resistance to the microtome knife, the resulting ragged artefact revealing nothing to show what may have been present. Moreover, in the examination of a relatively small number of sections, one or two, from each tonsil, those islands of cartilage and bone which are located in the more anterior and posterior portions of the capsule are necessarily missed. This is not as effective a source of error as might be imagined, for the tonsils having bone or cartilage in such situations usually

have multiple nodules elsewhere as well. Nevertheless, the inability to study serial sections in dealing with so extensive a material cannot do otherwise than produce subminimal values for incidence. The writer is fully aware of certain other sources of error inherent in dealing with material in which a large number of variable factors are simultaneously operative. It may well be that the presence of bone and cartilage in the tonsil favors those conditions which bring the patient to the otolaryngologist, or that, on the other hand, such conditions may have had some causal relationship to the production of the bone and cartilage. In that connection it must be remembered that the only claim made for the figures here set forth is that they represent a minimum incidence value for bone and cartilage in tonsils removed at operation. It is the belief of the writer that the incidence in the general population, if obtainable by the same method, would not be far different from the value here ascertained, a belief based upon the relatively slight manifestations of disease which have been deemed sufficient to warrant tonsillectomy.

INCIDENCE.

General Incidence.—The tonsils from 1,000 consecutive tonsillectomies examined histologically show cartilage or bone, or both, in 209 cases, an incidence of 20.9 per cent. Few similar series are offered for comparison in the literature. In 50 tonsils from 37 different individuals Reitmann²² found bone or cartilage in 17, or 34 per cent. Apparently, these were from 14 individuals, an incidence of 38 per cent on a basis similar to ours. In the material from 412 autopsies Lubarsch¹⁶ found bone and cartilage 65 times, or 15.77 per cent. Maclachlan¹⁷ collected eleven cases among 350 pairs of tonsils, an incidence of a little over 3 per cent. Forman⁷ found only one specimen containing cartilage in the examination of 290 consecutive pairs of tonsils. This remarkably small incidence may have been associated, he suggests, with an almost equally low percentage of tuberculosis. From our experience, we know that some other factor must have been operative, for the coincidence of those two conditions is no more frequent than chance association demands. It must be understood that for reasons which have already been given the incidence of 20.9 per cent determined by us is a minimum figure. Also, in any series,

the age distribution in the entire group will be found to greatly influence the incidence. Comparisons can be made profitably only when such factors are considered.

In the 209 cases positive for bone and cartilage, in the series here reported, 53 showed cartilage in both tonsils, while in 156 it was found in but one. Similarly of the 59 with bone, 19 showed bone in both tonsils and 40 in only one. The 59 cases in which bone was found are all included in the 209 showing cartilage. In other words, while bone was never found without some cartilage occurring also, it was found in but 28.3 per cent of the cases showing cartilage.

Incidence as Influenced by Sex.—In the 1,000 consecutive pairs of tonsils there were 579 from male patients and 421 from female patients. The former yielded 126 cases showing bone or cartilage, an incidence of 21.76 per cent. The 421 cases in females yielded 83 positive cases, an incidence of 19.71 per cent. The difference between the percentages of incidence in the two sexes is without significance, falling well within the range of error inherent in the method of selection and analysis of this material.

Incidence as Influenced by Age.—In the 1,000 consecutive tonsil cases of known age (a slightly different series than the one considered for general and sex incidence) 205 cases showed the presence of bone or cartilage or both. The youngest positive case was at age three, with three positive cases at age four and five positive cases at age five. The oldest positive case was found at age 59, with two at 56 and two at 55. Since this material was surgical in origin and not derived from the autopsy table, the extremes of life have but a meager representation. The incidence just given is therefore illustrative only of the fact that bone and cartilage may be met with at any age.

The average age of the 1,000 cases was 18.722 years, while the average age of the cases showing cartilage and bone was 24.263. That advancing years increase the incidence is still more evident when it is noted that the average age of the 795 negative cases was 17.295 years, making a difference of almost exactly seven years in the average ages of the two groups. Moreover, it must be borne in mind that those in the second, third and fourth quinary periods of life constitute the group

from which the larger proportion of the tonsillectomies is done. If consecutive cases are considered, this period, therefore, will have undue weight in determining the average age, provided the incidence of bone and cartilage is not fairly uniformly distributed throughout the years of life, and it is evident from the average ages of the positive and negative cases in this series that further analysis as to age distribution is indicated for the same reason.

If, then, the cases be segregated according to age periods and the percentage of bone and cartilage incidence be determined separately for each period, the following table can be constructed:

TABLE I.

Age Period	Total Cases	Positive	Negative	Incidence
1— 5	75	9	66	12%
6—10	236	23	213	10
11—15	187	20	167	11
16—20	133	32	101	32
21—25	120	30	90	33
26—30	90	34	56	61
31—40	96	35	61	57
41—73	63	22	41	54
	1,000	205	795	

The change of incidence is shown graphically in Figure 1, in which the values of the final column of the table are plotted as if the cases were concentrated in the terminal year of each age period. It will be noted that in spite of the large number of cases considered and the consequent reduction in error, the incidence of bone and cartilage remains practically constant for the first 15 years of life. It then rises rapidly in the next fifteen years to a level five times as high, where it remains without further increment, the slight downward tendency in the later years probably being an inaccuracy introduced by the greatly lessened number of cases.

HISTOPATHOLOGY.

Cartilage and bone are found only in the connective tissue structures of the tonsil, the capsule and the trabeculae. Apparent exceptions to this rule occur, in which islands of cartilage

are found close to the pharyngeal mucosa or partially included within lymphoid tissue. Careful examination will always show, however, that such islands of bone or cartilage are entirely included within extensions from the capsule and never within the actual substance of the lymphadenoid tissue. (See Figure 8.)

In searching for the occurrence of these substances it is noted that cartilage is more frequently found in association with the small mucous glands of the tonsillar capsule (Figures 2, 4 and 5), particularly the group occurring near the lower pole, than in any other situation. This finding is met with so frequently that the tonsils showing it constitute a special group of cases. In this location it occurs in even very young children, in whom it is by far the most frequent type and in whom it is oftentimes found unassociated with any increase in the general stroma of the tonsil. When thus found, it may be the only nodule of cartilage present. Groups of voluntary muscle fibers are frequently associated with the cartilage and mucous gland alveoli in this type, but it is very rare to find ossification in the islands of cartilage occurring in this situation.

With this group of cases can be contrasted another, occurring more frequently in older patients, although we have seen it a number of times in children under six years. In these there is no special association of the bone and cartilage with the mucous glands, but the association is with an increase of stroma and fibrosis of the supporting tissue. The islands of bone and cartilage are usually multiple (Figure 3), sometimes very numerous and tend gradually to coalesce, forming in extreme cases a ramifying skeletal mass occupying the thickened capsule and trabeculae. In this type ossification is very apt to occur.

Membranous osteogenesis has not been found in any instance and probably does not occur in the tonsil. Therefore bone is never encountered without cartilage being present also. However, there are several instances in our total series in which the rim of cartilage surrounding an island of bone is so narrow that it might be very easily overlooked if not closely examined. In such cases, the usual progression of connective tissue to cartilage and of cartilage to bone is apparently inter-

rupted, in that the new formation of cartilage practically ceases while bone formation continues until the cartilage is almost, if not quite, exhausted.

In those tonsils in which the supporting connective tissue contains numerous islands of cartilage and bone, all stages in the histogenesis of those tissues may be found at the same time. The process is progressive and continuous, leading from dense connective tissue, microscopically showing little to differentiate it from any other connective tissue, to bone with well developed Haversian canals and a highly vascularized bone marrow. The process may, however, be retarded or checked at any stage.

The connective tissue of the areas in which cartilage is to develop (except those islands of cartilage developing in association with mucous glands) would in most cases be considered to be increased in amount, so that in a descriptive diagnosis such expressions as "increased stroma" or "fibrosis" would be applied to such a tonsil. This connective tissue is, perhaps, somewhat more coarsely fibrillar and shows a slightly greater tendency to form whorls than that ordinarily found. In such an area a very small, rounded localized center appears in which the fibrillar structure is less evident and the eosin staining diminished in intensity. As these changes become more marked, the cells become more rounded, their cytoplasm clearer and the nuclei oval or spherical (Figures 6 and 7). Such cells come to lie in a matrix which is more clear and which, with nearly complete loss of evidence of fibrillæ, becomes somewhat mucinoid, staining bluish with hemalum. In such areas, now closely resembling cartilage, the cells come to have characteristic clear capsular spaces, but show a more uniform distribution and less grouping than is usually seen in cartilage in other situations. This indicates relatively little growth of the islands of cartilage through proliferation of their fully differentiated cells. In full accord with this is the evidence of peripheral extension, for the process of transformation of connective tissue into cartilage may at times be found in all stages in the surrounding zone. In other instances the surrounding connective tissue becomes somewhat compressed and laminated and forms a definite capsule or perichondrium for the cartilage nodule. While no vessels are found in the substance of the

cartilage, the connective tissue capsule may be well supplied. The formation of the cartilage is therefore a process of direct metaplasia.

Calcification of the cartilage is the first step toward bone formation. The cartilage cells usually become larger and more globose while lime salts are deposited in a network of delicate trabeculae around and between them. This deposit of lime salts in the matrix continues until a heavy and sometimes coarsely granular mass is formed, staining a deep purplish blue with hemalum and eosin (Figures 9—12). True bone is formed only with the destruction of this primary calcification by a vascular osteoclastic and osteoblastic tissue, which is derived in part, and perhaps entirely, from the surrounding capsular structures. The source and nature of the cells concerned in this process present the same difficulties of interpretation as in endochondral ossification elsewhere, a process which the further elaboration of true bone in the tonsil resembles in every particular. In a number of instances we have seen the usual convex surface of the nodule of partially calcified cartilage indented by a fibroblastic and angioblastic bud apparently invading it from the vascular capsule. This is apparently the method by which the osteoblastic tissue reaches the interior of the nodule. Then by repeated phases of formation and destruction the bony area enlarges peripherally, but soon comes to have a fully developed marrow space in the interior, which may contain a fatty, highly vascular marrow (Figures 13 and 14). The surrounding bone shows typical lacunae, leaving no room for the doubt expressed by Grove¹⁰ as to the occurrence of true bone in the tonsil.

Although we have seen many instances of the type of cartilage occurrence in which there is a solitary nodule or barlike mass in association with the mucous glands and without noteworthy increase in stroma, we have never observed the stages of bone formation in these cases. We must conclude that this cartilage differs in certain respects from that found in the other type and that one of these differences is its tendency to remain unaltered for many years, failing to show the primary calcification which in other situations is the forerunner of osteogenesis.

There is no fixed association of cartilage and bone in the

tonsil with tuberculosis or any other specific infection. We have now seen several hundred examples of tonsillar tuberculosis, and we find that such tonsils show cartilage and bone in approximately the same proportion as the nontuberculous tonsils of patients of the same age groups. There is a definite association of the type in which multiple nodules of cartilage and bone are found, with a well marked increase in the connective tissue of the capsule and trabeculae, but it is noteworthy that many cases with equally marked fibrosis show no bone or cartilage. Kellert¹³ concluded that there is no certain relation between cartilage in the tonsillar capsule and inflammation.

DISCUSSION.

In discussing the results of this analysis it will appear that they support the belief that the occurrence of bone and cartilage in the tonsil is not due to any misplacement of cartilage or bone forming cells or to any other disturbance of development in the sense in which that expression was used by the earlier writers upon this subject. The process is essentially one of metaplasia, but is made possible by the inherent nature of the tissue concerned and is furthered by the occurrence of a chronic fibrosing inflammatory reaction.

For the earlier arguments between those believing the process one of metaplasia and those insisting that such formations could come only from "rests," the papers of Rückert,²⁴ Carter,³ Theodore,²⁷ Cary,⁴ Albert and Douglass¹ and others may be consulted. Nösske,¹⁹ in 1903, had come the nearest to an accurate statement of the genesis of tonsillar bone and cartilage when he wrote that in addition to inflammatory and retrogressive processes a sort of local predisposition plays an etiologic rôle; that the connective tissue of the tonsil is endowed with a special cartilage and bone forming capacity which can depend upon a developmental "moment" without the necessity of assuming any embryologic disturbance.

In a recent paper Lipshutz¹⁵ emphasizes the importance of a thorough appreciation of the part played by the structures derived from the second branchial bar in connection with the occurrence of either continuous or discontinuous bony or cartilaginous masses along the course of the stylohyoid ligament. In its origin from the second branchial pouch the tonsil de-

rives its supporting tissue from the second and third branchial arches. Themselves cartilaginous, the branchial arches contribute a tissue which possesses to an unusual degree the power of cartilage and bone development. We find that the observed facts are in complete accord with this explanation. The tonsils of young children which show an incidence of but 10 per cent or 12 per cent of bone and cartilage, if allowed to remain in position twenty years or so longer would show an incidence of bone and cartilage of 50 to 60 per cent. Yet one cannot tell by microscopic examination which of the tonsils of very young children would, and which would not, produce cartilage and bone if left in. There are no "rests" of cartilage or bone cells. Moreover, the inherent power to form cartilage and bone is not possessed by the tonsillar supporting tissue to the same degree in all cases. Some tonsils show marked fibrosis without any cartilage.

The method of osteogenesis in the tonsil is that which would be expected in derivatives from the branchial bars. There is no authentic evidence of intramembranous ossification in the tonsil, and we believe that the instances in which bone seemed to be present without cartilage are to be explained by a complete or nearly complete replacement of the cartilage by bone without the continuous production of new cartilage at the periphery of the nodule, as is usually the case.

The occurrence of nodules of cartilage at the site of the developing tonsil in fetuses, as described by Lubarsch¹⁶ and others, by no means militates against the view here presented. In the first place, there is a legitimate doubt as to the exact location and relationship of the nodules found in such fetuses, due to the relatively late development of the tonsil itself. Even if found at an early age, such nodules are still subject to the same explanations as in later life, for the cartilage forming potentiality may become effective at any age.

Is the difference between an incidence of 10 per cent at 10 years of age and an incidence of 60 per cent two decades later to be explained by the one fact alone that time has elapsed for the development of cartilage and bone nodules in a tissue having that potentiality? It scarcely seems possible that so abrupt a change could result if this were the sole factor. Rather it would seem that the proliferation of new connective tissue

associated with the healing of repeated inflammatory processes itself awakens and stimulates the latent power. One is forced to this conclusion by the frequent association of much scar tissue and a general increase of connective tissue with multiple nodules of bone and cartilage in the very young as well as in the old. That cases occur without much or even any diffuse fibrosis serves but to demonstrate more completely the importance of the histogenetic inheritance of this tissue. Particularly is it true in the type of case in which a single bar of cartilage is found in association with the mucous gland and often also with a few voluntary muscle fibers that chronic inflammation plays an insignificant part. This fact is in accord with the observation that this is the more common type in very young children. It must be true that the supporting tissue of this area is prone to produce cartilage without the aid of stimuli dependent upon or associated with any known pathologic condition.

CONCLUSIONS.

1. In 1,000 consecutive pairs of tonsils, out of 15,000 examined, the minimum incidence of cartilage and bone, as determined by the systematic microscopic search of a limited number of sections from each tonsil, was found to be 20.9 per cent.

2. Bone was never found without at least some cartilage and occurred in 28.3 per cent of the 209 cases in which cartilage was found.

3. The incidence was not significantly affected by a segregation of the cases as to sex.

4. The average age of cases showing cartilage or bone was 24.263 years; of those which were negative, 17.295 years.

5. The incidence of cartilage and bone remains at a nearly constant level up to puberty. It then rises rapidly to a high level at age period 26 to 30, after which it appears to remain practically constant. The period of most rapid increase in incidence is thus coincident with the period of most active osteogenesis in the skeletal development of the individual and is therefore apparently subject to the same laws as those governing general bone development.

6. Cartilage and bone are found only in the connective tissue structures of the tonsil, where they occur in two somewhat distinct types.

7. In one type the microscopic sections show a single mass of cartilage in close association with a mucous gland. We have not observed bone formation in this type. It is possible that the occurrence of cartilage in this situation is within the range of anatomic variation and has no pathologic significance.

8. In another type multiple foci of cartilage and bone occur throughout the capsule and trabeculae, usually associated with considerable increase in connective tissue.

9. Cartilage is formed by a process of progressive transformation (direct metaplasia) of the connective tissue, while bone in the tonsil always arises in such newly formed cartilage by processes wholly comparable to endochondral ossification elsewhere.

10. Disturbance of development in the sense of "rests" of cartilage and bone cells cannot explain the occurrence of these tissues in the tonsil.

11. The inherent predisposition of the connective tissue of the tonsil to form cartilage depends upon its origin from branchial arch structures. This latent tendency becomes especially effective under the influence of chronic inflammation.

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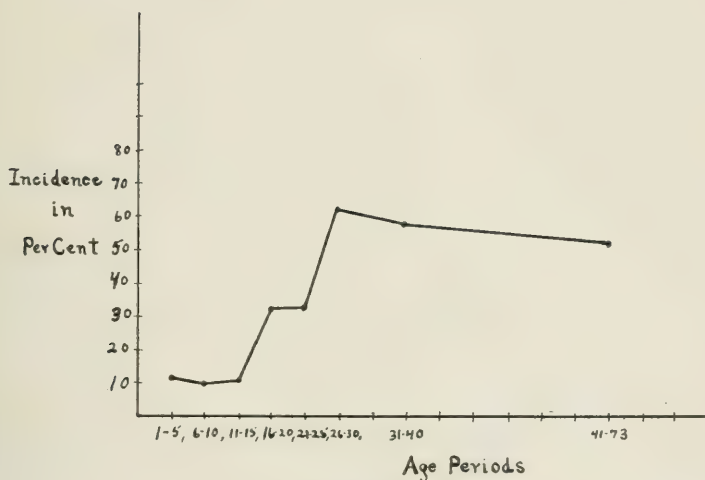


FIGURE 1.

Curve of age-incidence of bone and cartilage in the tonsil, based upon 205 consecutive positive cases of known age. The percentage of incidence is plotted against five year age periods. The maximum incidence is reached in the age of 26 to 30, after which there is practically no change.

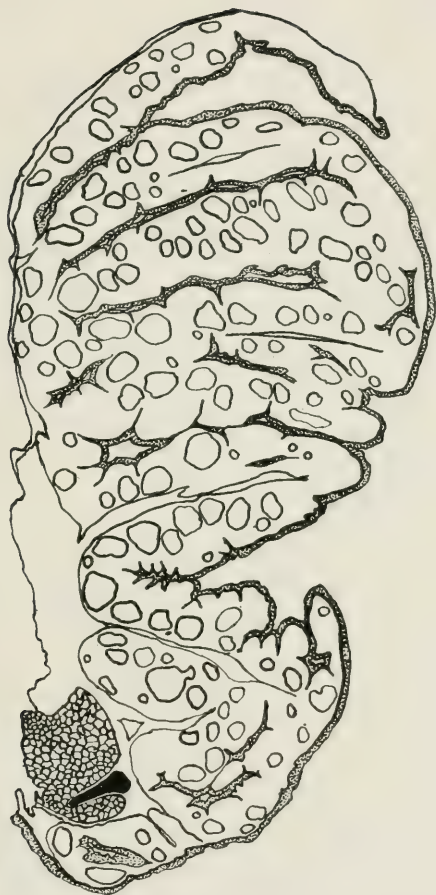


FIGURE 2.

Outline tracing of actual section of tonsil cut through the greatest vertical plane, to show the location of the cartilage (in solid black) as very frequently found near the mucous glands. The more frequent type in young children and unassociated with increase in stroma. This is probably a normally occurring structure in many individuals.

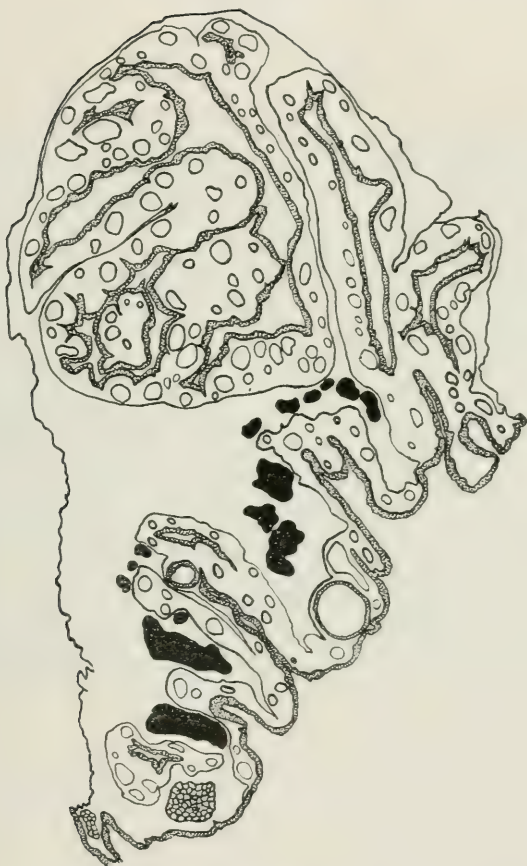


FIGURE 3.

Outline tracing of actual section of tonsil to show the distribution of bone and cartilage (solid black) in the group of cases in which the association is not with the mucous glands but with increase of stroma and fibrosis of the supporting tissue.

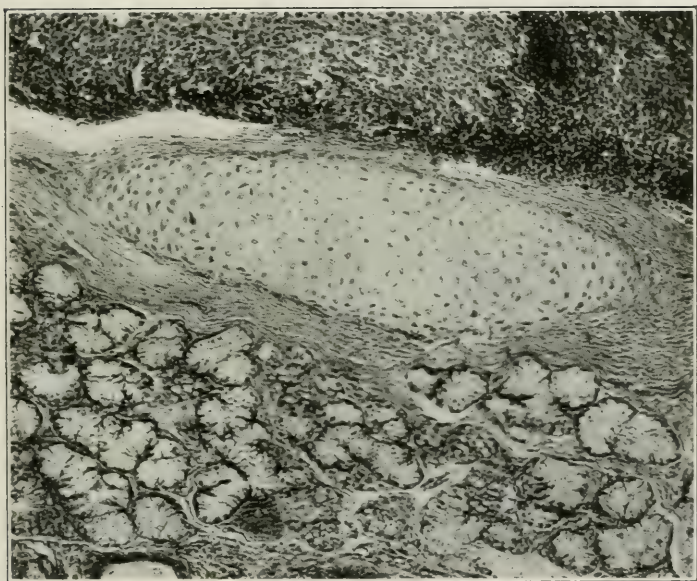


FIGURE 4.

Photomicrograph (hemalum and eosin stain, Zeiss obj. "B," no ocular) showing nodule of cartilage occurring in association with mucous gland alveoli. The more frequent type in young children.



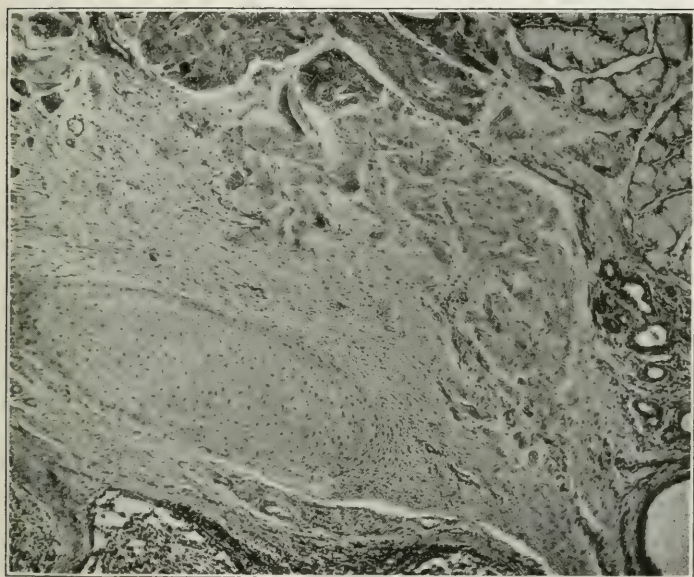


FIGURE 5.

Photomicrograph showing nodule of cartilage occurring in association with mucous glands and a group of voluntary muscle fibers. Another example of the type first described in the text.

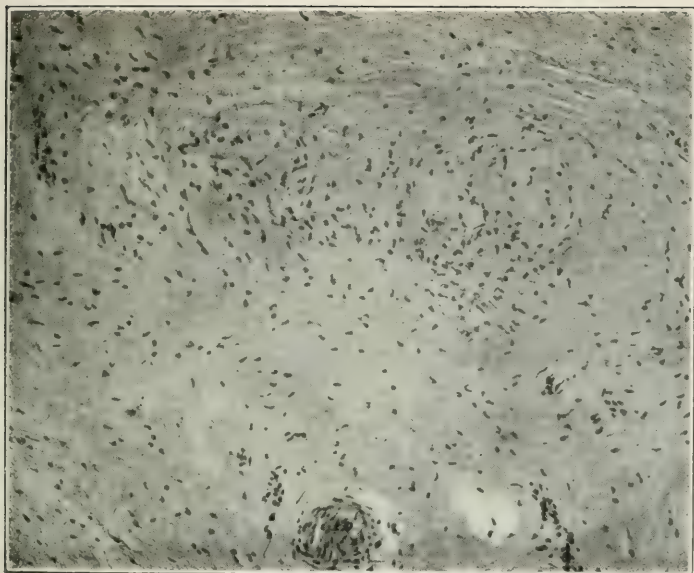


FIGURE 6.

Photomicrograph showing an early stage in the direct metaplasia of fibrous connective tissue to cartilage in the stroma of the tonsil. This tonsil showed the simultaneous development of multiple cartilage nodules. These nodules show no relationship to the normally occurring mucous glands. The type more commonly found in adult patients.



FIGURE 7.

Photomicrograph of somewhat more advanced cartilage formation than that shown in Figure 6. Many of the cells have now become globular and the cartilage character of the nodule is well established.

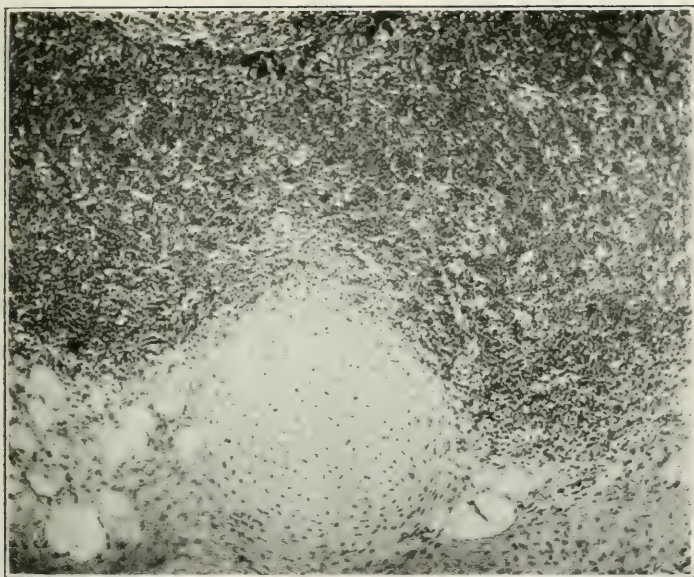


FIGURE 8.

Photomicrograph showing a well developed nodule of cartilage in the stroma of the tonsil. It is so close to the lymphoid tissue that it intrudes upon it, although actually situated entirely in the stroma. This appearance has at times been incorrectly interpreted as cartilage developing in the lymphoid tissue.

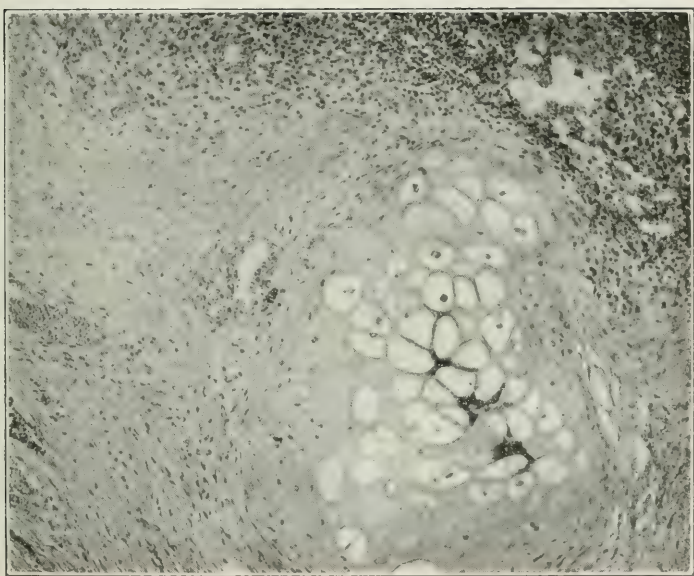


FIGURE 9.

Photomicrograph showing early stage in the calcification of cartilage in the tonsil. The first step toward bone formation.

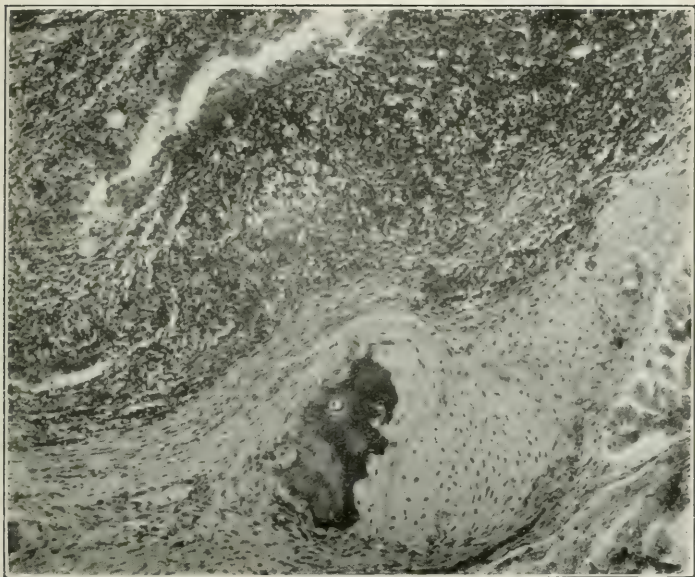


FIGURE 10.

Photomicrograph of more advanced calcification of the cartilage in the tonsil.

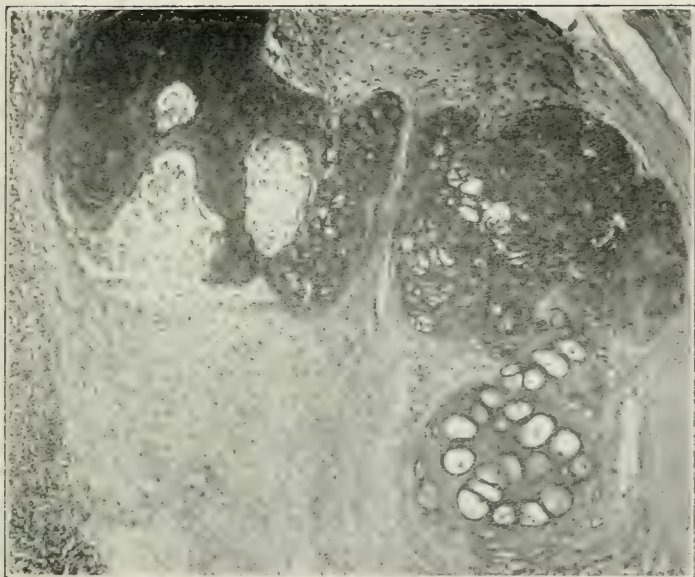


FIGURE 11.

Photomicrograph showing in a single field several stages of transformation of cartilage into bone. Early and later stages of calcification of cartilage, true bone formation, ingrowth of vascular tissue to form the marrow.

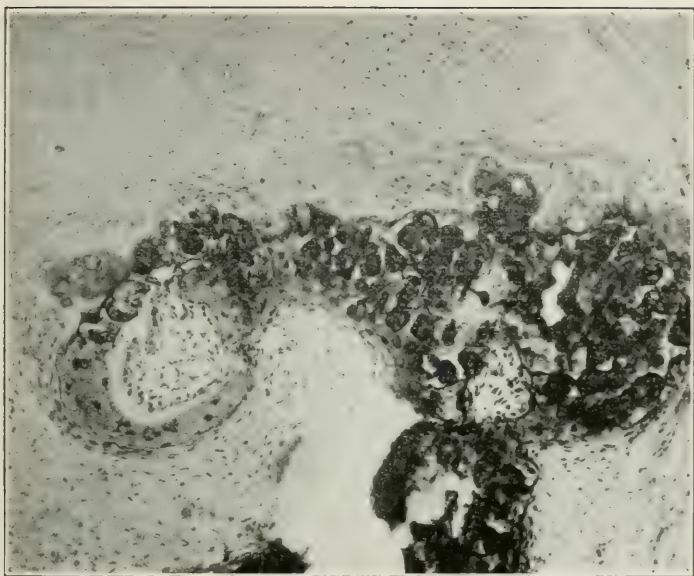


FIGURE 12.

Irregular calcification of cartilage and replacement of the calcified cartilage by true bone with included bone cells and a vascular marrow.

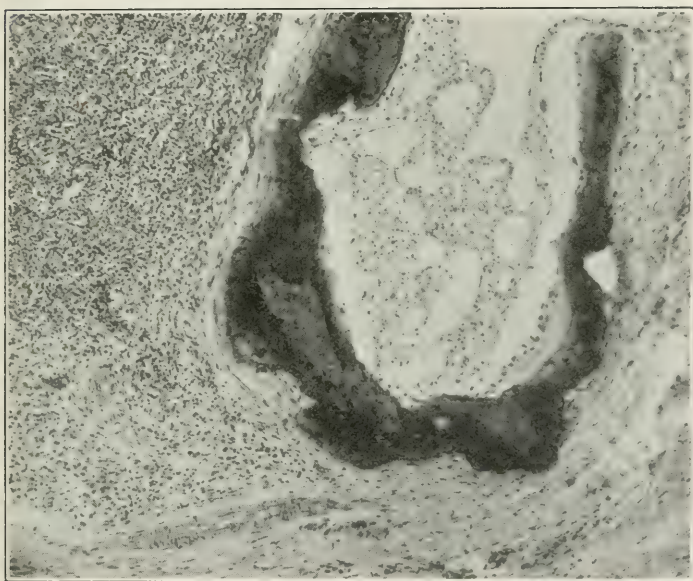


FIGURE 13.

Photomicrograph of advanced bone formation in the tonsil. Bone cells and a very abundant marrow are present. Some of the calcified cartilage still persists.

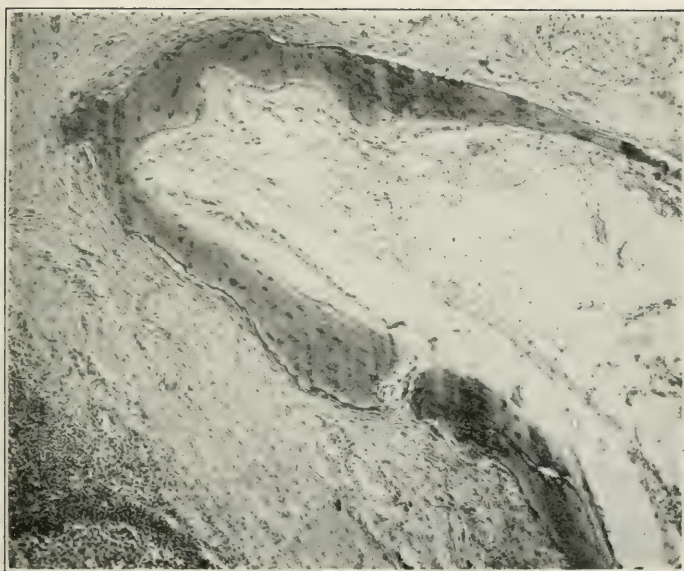


FIGURE 14.

Photomicrograph of advanced bone formation. Process now practically complete. Typical bone cells and abundant vascular and fatty marrow. Canal through bony wall for entrance of vessels to the marrow and endosteum.

LI.

NOTE ON THE RELATION OF THE OPTIC NERVE TO THE LAST POSTERIOR ETHMOID CELL.*

BY S. B. WESTLAKE, M. D.,

ST. LOUIS.

The clinical relations of the eye to the nose have been made the subject of much study in the last few years. The variety of these clinical manifestations have been almost endless; in fact, it would appear that almost every eye affection has been attributed to a nasal condition. We are, however, wandering away from reflex action as responsible for many of these conditions and are searching for definite pathologic etiology. The sinus relations have been the basis of most of the investigation of this subject, whether it be anatomic, physiologic or bacteriologic. When these have been considered inadequate by way of explanation, focal infection has been held accountable.

But it is being recognized that if we are to achieve any great success in this study, and if we are to have a definite pathologic basis, we must have more essential anatomic data.

The value of definiteness in this respect was beautifully shown by Van der Hoeve at the meeting of the American Academy of Ophthalmology and Otolaryngology in September, 1921, in the report of a case. The symptoms in this patient had persisted in spite of extensive operation. It was only after an ethmoid cell had been located by a careful X-ray examination and exenterated, that the eye symptoms entirely disappeared.

Further studies in the anatomy of the paranasal sinuses are therefore necessary.

*From the Department of Anatomy and of Ear, Nose and Throat Diseases, St. Louis University.

H. W. Loeb ("A Study of the Anatomic Relations of the Optic Nerve to the Accessory Cavities of the Nose," *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, St. Louis, 1909) in his investigation on the relation of the optic nerve to the paranasal sinuses, made some dissections which showed that the optic nerve usually has a minimal relation to the ethmoid labyrinth.

His series of fifteen heads sectioned horizontally showed that in 28 of the 30 instances the optic nerve came into close relation with the last posterior ethmoid cell (occasionally there were two) only at the posterior external superior angle of the cell, from which point it left the labyrinth region to attach itself to the globe. In two instances, however, where the sphenoid was replaced by an ethmoid cell, the nerve's course was along the external wall of the cell for a considerable distance.

To continue these studies with a view of confirming or disproving his observations, he suggested that I should prepare new dissections which would make it possible to study the external cranial wall of the ethmoid cells and sphenoid sinuses. Accordingly seven heads were decalcified and treated according to the technic described by him and, after the cranial vault above the level of the sella had been cut away and the brain removed, the superior and external bony walls of the sinuses were cut away, as far as possible maintaining the mucosa intact and the optic nerve prepared from the periorbita. The results are as follows:

Head I (Fig. 1).—Both sphenoids, fairly large and uniform in size, shown cut open, with mucosa lost. Ethmoid cells on the left side shown with mucosa of external wall intact, that of the roof being cut away. Optic nerve courses along the external wall of the sphenoid, just below its roof, and the superior posterior external angle of the last posterior ethmoid cell from which it proceeds externally to the globe.

Head II (Fig. 2).—The optic nerve has been cut away, exposing the mucosa of the maxillary roof and the external walls of the ethmoid cells and sphenoid sinus.

Head III (Fig. 3) (Right).—This most successful preparation shows the complete mucosa of the superior and external wall of the maxillary sinus and of the external walls of the ethmoid cells. The mucosa of the sphenoid was lost in the preparation, but the bone cavity itself is shown in relation with the optic nerve and adjacent ethmoid cells. The optic nerve presents the usual relation with the last posterior ethmoid at its posterior, superior and external angle.

Head III (Fig. 4) (Left).—In this preparation the dissection was not so extensive as that of the right side, a portion of the bony wall being retained to establish its relation to the optic nerve.

Head IV (Fig. 5).—Only the ethmoid cells have been prepared with their mucosa intact. Usual relation.

Head V (Fig. 6).—Small opening in the sphenoid roof is seen and also in the last posterior ethmoid cells, the mucosa of the external wall of the left side being retained. The usual relation of the optic nerve and last posterior ethmoid cell is definitely shown.

Head VI (Fig. 7).—In this, the relation of the optic chiasm to the two sphenoids is well shown and also the usual relation of the nerves to the right and left last posterior ethmoid cells.

Head VII (Fig. 8).—The left sphenoid is observed in relation with the chiasm and both optic nerves. The mucosa of the right sphenoid is seen projecting upwards into the last posterior ethmoid cells on that side, being separated from the optic nerve by the wall and a portion of the cavity of the ethmoid cell. The right optic nerve courses along the external wall of this cell for at least 1.5 centimeters, corresponding to the observation made by Loeb.

With this confirmation increased emphasis is given to the claim made that the optic nerve becomes more vulnerable to infection of sinus origin whenever through replacement of the sphenoid cavity it runs in close proximity to the outer wall of the last ethmoid cell.

Designating one the angular relation and the unusual form, the mural relation, the results of the study of the twenty-three heads are as follows:

	Angular	Per Cent	Mural	Per Cent
Loeb's series	28	93.3	2	6.7
Present series	15	93.8	1	6.2
	—	—	—	—
Total.....	43	93.5	3	6.5

The remarkable agreement of these findings suggests further investigation along these lines.

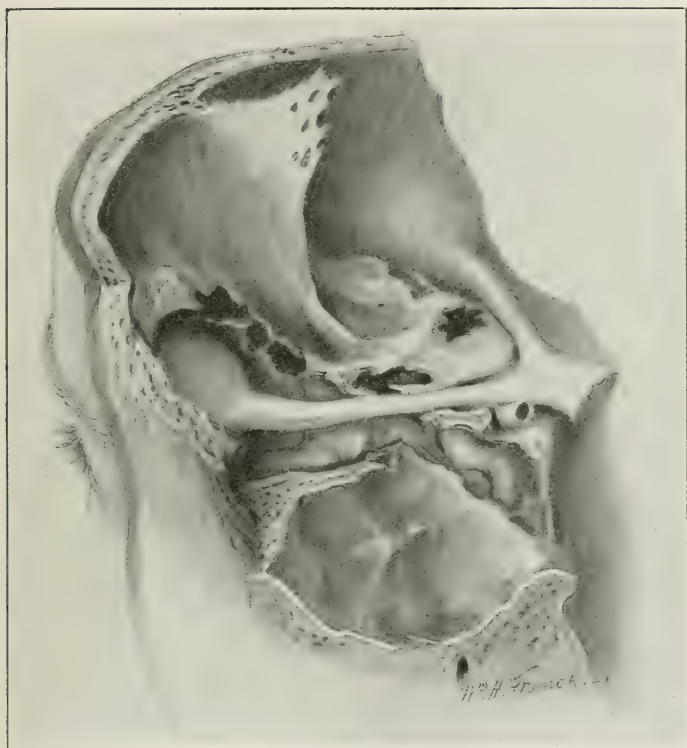


Figure 1.

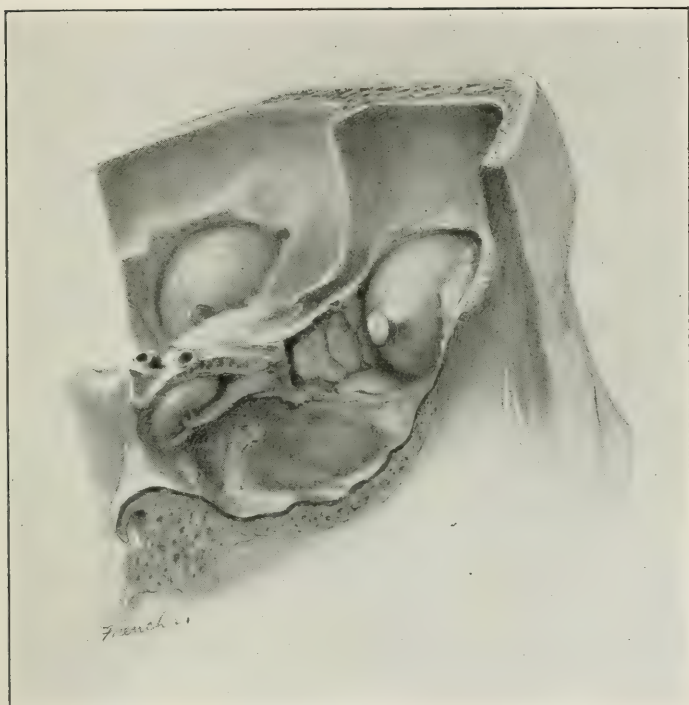


Figure 2.

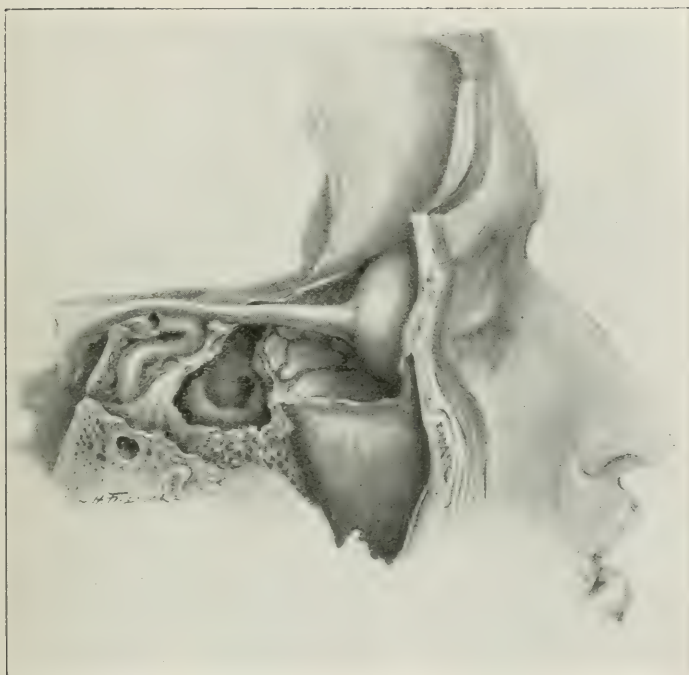


Figure 3.

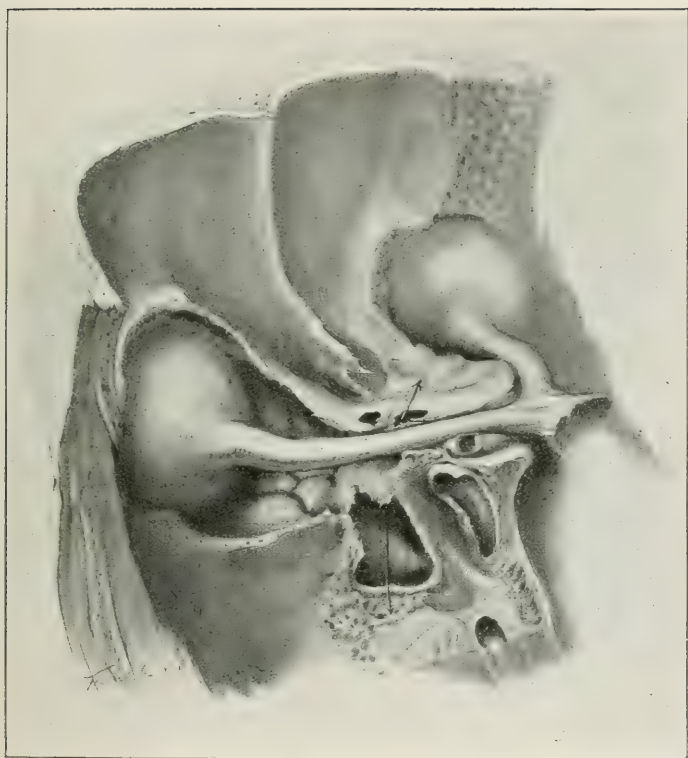


Figure 4.

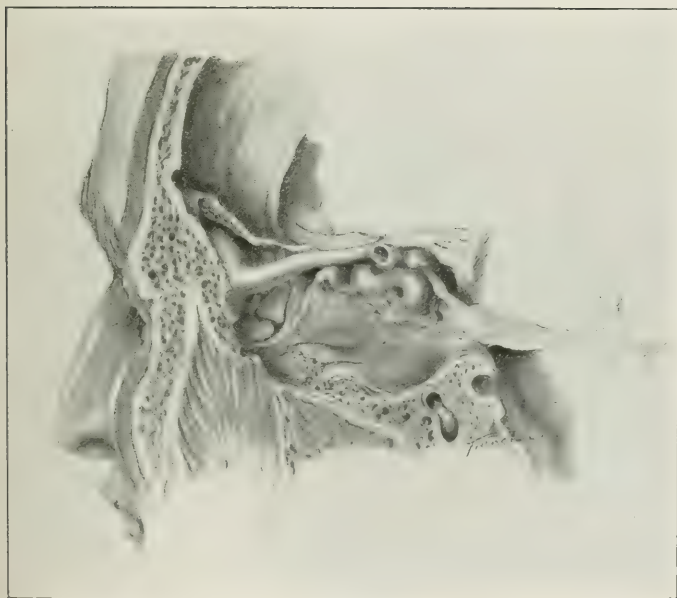


Figure 5.

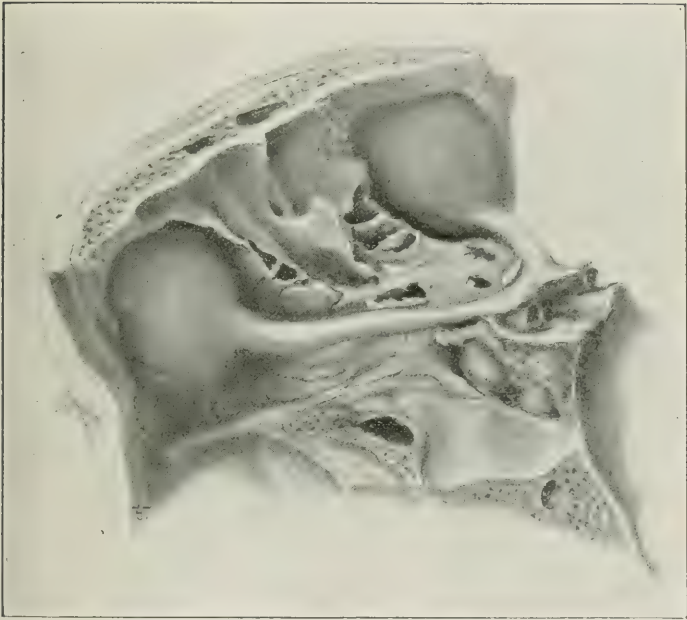


Figure 6.



Figure 7.



Figure 8.

LII.

A STUDY OF THE MECHANISM OF PAIN AS SEEN IN RHINOLOGIC AND LARYNGOLOGIC CASES.

By L. HUBERT, M. D.,

ASSISTANT SURGEON, MANHATTAN EYE, EAR AND THROAT
HOSPITAL,

NEW YORK.

Few problems in rhinology and laryngology are more difficult and involved than those centering about the interpretation of pain. The purpose of this paper is to explain briefly its mechanism. In order to understand this properly, it is well to classify pain into three general types—first, somatic; second, autonomic, and third, psychic pain, as the mode of production of these three types differs.

MECHANISM OF SOMATIC PAIN.

Somatic pain is due to irritation of the sensory nerve endings (pain sense organs) and of the pain nerve fibers. The latter are afferent somatic neurons, which have their cell bodies in the sensory, posterior root ganglia and pass into the central nervous system by the posterior nerve roots. The pain impulses, transmitted along the afferent somatic neurons and the pain tract within the central nervous system, finally reach the optic thalamus (containing the pain center) and the cerebrum. The latter becomes conscious of the stimulus and recognizes the place where it arose.

MECHANISM OF AUTONOMIC OR VISCERAL PAIN.

Autonomic pain is due to irritation of afferent autonomic nerve fibers of the viscera or of the structures that are visceral in origin—i. e., those that are developed embryologically from the branchial arches and pouches. These afferent autonomic nerve fibers have their cell bodies, like the afferent somatic fibers, in the posterior root ganglia (somatic sensory).¹ The pain impulses are, therefore, transmitted to some of these

sensory ganglionic cells and pain arises. The cerebrum recognizes this pain and forms a judgment of its locality. That locality, however, is not the region where the pain stimulus arose, but in the peripheral distribution of the somatic sensory nerves, whose cells in the posterior root ganglia had been stimulated. This is why autonomic pain is also called referred pain.

MECHANISM OF PSYCHIC PAIN.

Psychic pain originates in the cerebrum of neurotic persons and from there is projected to the periphery. The location and the character of this pain are influenced by the ideas the patients have about it. It is described as dull, sharp, constant or "coming and going," burning, twisting, scratching, etc.

Neurotic persons do not distinguish between discomfort and pain. Their nervous system seems to be so sensitized that the slightest discomfort makes them complain of severe pain.

The most important characteristics of psychic pain are the facts that it usually does not interfere with sleep and that superficial contacts, like combing the hair, are very painful, while deeper pressure may cause no discomfort.

PAIN OF NASAL ORIGIN.

The interpretation of pain as a result of nasal disorders is rather difficult. It is due to the complexity of the nasal innervation. The nose is not only supplied by a nerve of special sense, but also by somatic sensory and most probably by afferent autonomic nerves. It may, therefore, be considered not only as an organ of special sense but also as a somatic and visceral organ. We know how difficult it is at times to interpret pain caused by somatic sensory disturbances alone. The difficulty is still greater when we try to interpret pain caused by autonomic or visceral disturbances alone. The nose is an organ in which disease may produce both types of pain, either singly or combined. Besides, many neurotic patients suffering from psychic pain may show nasal abnormalities which are not the cause of the pain. Therefore, in our specialty, we may encounter the three types of pain previously mentioned, namely: (1) Somatic, (2) autonomic, (3) psychic. Before discussing in what manner the various nasal diseases may cause

pain it is well to describe the nerve supply of the nose in a little more detail than is usually done.

NERVE OF SPECIAL SENSATION.

The nerve of the special sense of smell is the olfactory or first cranial nerve. It has nothing to do with pain. Irritation of this nerve, however, may produce a disagreeable as well as an agreeable sensation. I know of a very sensitive patient who complains of getting a "headache" whenever she smells certain odors.

SOMATIC SENSORY INNERVATION OF THE NOSE.

The first and second division of the fifth cranial nerve constitute the somatic sensory innervation of the nose. According to those modern anatomists and physiologists² who have given this subject their attention, the sphenopalatine ganglion does not supply the nose with sensation, as is stated in all text-books of rhinology. The so-called sensory branches of this ganglion are continuations of the branches of the second division of the fifth cranial nerve. Bearing this in mind, let us restate the sensory supply of the nose.

OUTER NOSE.

Side of the nose, upper part; infratrochlear branch of the nasociliary nerve of the first division of the fifth cranial nerve. Side of the nose, lower part; nasal nerve of the infraorbital nerve of the second division. Tip and bridge of the nose; external nasal branch of the nasociliary nerve of the first division.

INNERVATION OF THE NASAL CAVITIES.

Septum, anterior part: anterior ethmoidal nerve (internal branch of nasociliary nerve); middle part: nasopalatine nerve of second division; posterior part: posterior superior nasal branches of the second division.

Outer wall, in front of the turbinates: anterior ethmoidal nerve (external branch of nasociliary).

Superior turbinate, superior meatus, middle turbinate: posterior superior nasal branches of the second division.

Middle meatus, inferior turbinate, inferior meatus: inferior nasal branch of large or anterior palatine nerve of second division. The anterior part of the inferior meatus receives

also a branch of the anterior superior dental nerve of the second division.

THE ACCESSORY SINUSES.

Antrum of Highmore: branches of the superior dental nerves of the second division.

Frontal and anterior ethmoidal sinuses: fibers of the nasociliary of the first division have been traced by Meckel and Langenbeck.³

Posterior ethmoidal and sphenoidal sinuses: posterior superior nasal branches of the second division; a sphenothmoidal branch of the nasociliary nerve has been described by Luschka, which passes through the posterior ethmoidal foramen and is distributed to the sphenoidal sinus and the posterior ethmoidal cells in front of the body of the sphenoid; orbital branches of the second division, according to Luschka, supply, besides the periosteum of the orbit, also the periosteum and mucous membrane of the posterior ethmoidal cells and sphenoid sinus.

NASAL SOMATIC PAIN.

Nasal somatic pain may be caused by disorders affecting (a) the peripheral nerve endings (pain sense organs); (b) the trunks of any of the above mentioned nerves. Let us trace how the pain impulse from the nose reaches consciousness. It starts in the peripheral nerve endings (pain sense organs), which end "free" between the epithelial cells of the mucous membrane⁴ of the nose and accessory sinuses. It may also begin in the nerve fibers themselves. It passes along these fibers to the Gasserian ganglion, where the cell bodies of all the somatic sensory nerves of the nose are located. From these cells central processes are given off which enter the pons and bifurcate into ascending and descending branches (roots). They end in the sensory nucleus of the fifth nerve. Here the neurons of the first order in the sensory pathway from the nose to the cerebrum stop and those of the second order begin. The latter pass from the cells of the sensory nucleus to the upper part of the pons, where most of them cross to the opposite side and form the trigeminothalamic tract. They end in a special portion of the optic thalamus, which is considered to be the

center of organic pain. From here impulses are transmitted over neurons of the third order to the sensory area of the cerebral cortex.

NASAL AUTONOMIC PAIN.

The nose, as mentioned above, can also be considered as a visceral organ. Like other such organs it is supplied by both the sympathetic and the parasympathetic nervous system. It is known that these systems furnish the nose with vasomotor and secretory motor nerves.⁵ The important question: "Do afferent autonomic fibers exist in this locality?" as in the thoracolumbar region, has not yet been definitely decided. There is clinical evidence, however, that such may be the case. There are cases on record where the Gasserian ganglion had been extirpated or the sensory root of the fifth nerve avulsed and yet pain was still present. Also pain referred to regions far removed from the distribution of the fifth cranial nerve occurs, and its explanation must be sought in a disturbance of afferent autonomic fibers. There is another related fact that would make us surmise that such fibers may be present within the nose. It is known that in the thoracic and lumbar region the white communicating rami contain afferent autonomic fibers which pass to the somatic sensory ganglia, where they have their cell bodies. The vidian nerve has also been considered as a white communicating ramus, which is formed by the large superficial petrosal nerve. It may, therefore, be assumed that it also contains afferent autonomic fibers. If it is true that afferent autonomic fibers are present within the nose, their probable course would be as follows: Accompanying the intranasal branches of the second division of the fifth cranial nerve, they pass through the sphenopalatine ganglion to the vidian nerve, and have their cell bodies probably in the geniculate ganglion, and possibly also in some of the cervical and upper thoracic ganglia. The geniculate ganglion gives off a branch to the middle ear, and so we can explain the occurrence of otalgia of nasal origin. From the cervical and upper thoracic ganglia fibers are given off which make up the sensory supply of the mastoid region, the occiput, the neck, the shoulder, the arm, the forearm, the hands and the fingers. Thus we are able to explain the pain in these regions as a re-

sult of irritation of the afferent autonomic fibers within the nose.

Let us trace now how an autonomic pain impulse from the nasal cavity reaches consciousness. It arises in the afferent autonomic nerve fibers, if it is true that such fibers are present within the nose. It passes along these fibers, which have their course within the nasal sensory branches of the second division of the fifth cranial nerve, through the sphenopalatine ganglion to the vidian nerve. From here it is transmitted through the large superficial petrosal nerve to the geniculate ganglion, and probably through the carotid plexus and the communicating rami to the cervical and upper thoracic ganglia. From these sensory ganglia it travels along the somatic sensory pathway to the optic thalamus and cerebrum. When it reaches the cerebrum the pain is referred along the peripheral distribution of those somatic sensory nerves whose cells in the geniculate, cervical and upper thoracic ganglia had been stimulated by it.

It is obvious that we cannot have nasal autonomic pain unless afferent autonomic fibers are present within the nose. It is also obvious that these fibers can be irritated in any part of their course, near the sphenopalatine ganglion or away from it. The term "sphenopalatine ganglion neurosis," as used by Sluder, would make one believe that the irritation of these afferent autonomic fibers can only occur at this ganglion, which I think we have no reason to assume, or that the pain is due to irritation of the ganglion itself. The fact that the application of cocain to the region of the sphenopalatine ganglion relieves all or most of the pains may be due to the circumstance that near this region we can get at practically the entire sensory innervation of the nose which is blocked by the cocain. It is also possible that this drug may anesthetize the afferent autonomic fibers, although, as far as I know, its effect on such fibers has never been demonstrated. The sphenopalatine ganglion, being an autonomic ganglion, has nothing to do with pain, except possibly with the causation of pain in the throat. Winkler⁶ is the only neuroanatomist of note who believes that this ganglion contains a few sensory cells, which give off fibers to the soft palate but none to the nose. This is why it is possible to have pain in the throat in disease around the sphenopalatine ganglion.

What is the difference, if any, between autonomic and somatic pain? It is obviously difficult to determine whether the pain impulses from the nose reach the central nervous system by way of the afferent somatic or the afferent autonomic neurons, as the peripheral processes of the latter use the former as a pathway. It is difficult to conceive that the afferent autonomic fibers are irritated without simultaneous irritation of the afferent somatic fibers, unless we assume that the irritant is of such a nature that it would affect one set of fibers only, which may be possible. It is most probable, however, that both systems of nerves may become involved and that most of the pains are due to involvement of the somatic sensory system. It is impossible to distinguish from the character of the pain whether it is of autonomic or somatic origin. But when the pain is referred to regions far removed from the distribution of the terminal filaments of the fifth cranial nerve it may be considered as referred pain of autonomic origin. Associated symptoms, like sudden pallor or sudden flushing, salivations, etc., may also help us in deciding whether the pain is of autonomic origin or not.

NASAL DISEASES THAT CAUSE PAIN.

1. Suppurative Diseases of the Outer Nose.—Among the suppurative diseases of the outer nose that cause severe pain the following are the most frequent: Furunculosis, folliculitis, cellulitis and erysipelas. A folliculitis is quite frequently overlooked. It is necessary to inspect carefully the entrance of the nose to find the lesion. The pain in these diseases is due to irritation of the nerve endings or nerve fibers of either the nasal branch of the infraorbital nerve of the second division or of the external nasal and infratrochlear branches of the nasociliary of the first division, depending upon the location of the lesions.

2.—Septal Abscess.—A septal abscess is frequently accompanied by severe pain. It is due to irritation of the nerve endings or nerve filaments of the anterior ethmoidal nerve, a branch of the nasociliary nerve.

3. Pressure of Turbinates on Septum.—Many observers have described pain due to contact between the septum and the turbinates. It is supposed that in these cases inflammatory

conditions in the nasal sinuses are absent and that relief is obtained by shrinking the mucous membrane of the turbinates and the septum in contact with it, by means of cocain or adrenalin, or by operation. However, the frequent occurrences of anatomic variations in the nasal cavities, producing contact points between the turbinates and the septum, without pain, makes one doubt whether it is ever caused in this way. It is most probably due to an obscure nasal sinusitis, which is the cause of the congested and enlarged turbinates.

4. Vacuum Pain.—Vacuum pain is supposed to be due to nondiseased and obstructed sinuses. Readers are referred to Sluder's work for the explanation of its mechanism.⁷

5. Acute Nasal Sinusitis.—Affections of the accessory sinuses of the nose are the disorders par excellence that produce pain. In the acute cases there are two forms of pain: first, a constant pain, localized more or less over the diseased sinus, and second, a paroxysmal pain, neuralgic in character.

The constant pain over the sinus can be explained by the involvement of the very sensitive periosteum of its wall. It is also possible that at the same time with the inflammatory changes of the mucous membrane of the sinus its sensory nerve filaments also suffer inflammatory changes. In the latter instance a neuritis is probably present. It is known that in neuritis the pain is usually constant, and its location corresponds to the distribution of the nerve involved. Therefore, a knowledge of the sensory innervation of the diseased sinus will help us to a certain extent in the diagnosis. The paroxysmal pain, however, does not by its location indicate the involvement of a particular sinus. This pain is most probably caused by a neuralgia of the irritated nerve fibers of the diseased sinus. In any neuralgic condition we know that the pain does not correspond to the distribution of the particular nerve involved, but it appears in certain rather ill defined zones and areas corresponding roughly to the different trunks of the main nerve.

THE TIME OF APPEARANCE OF THE PAROXYSMAL PAIN.

The paroxysmal pain, which may result from the involvement of any of the nasal sinuses, is characterized by its appearance at a certain definite hour of the day. The opinion

that it is due to periodic increased secretion of pus is not based on accurate observation. A sinus may be full of pus without causing any pain. Of course, this does not mean that an empyema of a nasal sinus does not produce pain but only that it does not explain its periodic appearance. It is possible, however, that the habits of the particular patient have something to do with it. It is known that certain factors which increase blood pressure, like smoking, alcohol, stooping, also increase the pain. It is, moreover, recognized that the blood pressure undergoes variations during the twenty-four hours of the day. Faught⁸ calls attention to the ever-varying conditions, both internal and external to the body, that are continually at work and result in a varying blood pressure picture. Janeway⁹ has reported daily systolic variations amounting to 60 mm. of mercury. We may, therefore, assume that changes in the circulation of the blood which increase blood pressure have an influence on the appearance of the pain.

When a patient gives a history of pain occurring at a particular time, we should inquire as to his habits of life, his routine of work, how and when he eats. The blood pressure should be taken at frequent intervals during the day.

6. Chronic Nasal Sinusitis.—In chronic sinusitis pain is usually absent. If present, it is frequently characterized by a diffuse headache, which is probably due to toxic absorption from the infected sinus or sinuses. Patients suffering from chronic sinusitis are also subject to acute exacerbations, during which time the pain assumes the character of an acute sinusitis. There are patients with chronic sinusitis that suffer very severe pain, even without acute attacks. It is supposed to be due to an osteitis of the nasal sinuses, which causes pressure on the sensory nerve filaments. There are undoubtedly patients, however, who have a complete osteitis of the ethmoid and sphenoid sinuses, as may be demonstrated during an operation, and yet they do not complain of any pain. There must, therefore, be some other factors besides the osteitis that would explain this severe pain. It is possible that it is due to a disease of the nerve fibers themselves. In order to demonstrate this it would be necessary to make careful sections of the operated material from the nasal sinuses and prepare these sections with the special

stains for nervous tissue. This is a very tedious and difficult procedure and pathologists are not anxious to do it for us.

It is important to keep in mind that patients suffering from chronic sinusitis without pain may develop this symptom subsequent to an operation on the sinuses. This should be a warning to those who consider minor and annoying nasal symptoms as sufficient indications for this very serious operation.

CONSIDERATION OF THE SEPARATE SINUSES.

Acute Maxillary Sinusitis.—In acute maxillary sinusitis the pain may be either constant and localized over the antrum (periosteal and neuritic), or neuralgic in character, affecting the terminal filaments of the posterior superior, middle superior and anterior superior dental nerves of the second division of the fifth cranial nerve. The pain radiates, therefore, to the incisor, canine, bicuspid and molar teeth, also along the infraorbital nerve to the lower eyelid, side of the nose, cheek and upper lip.

Chronic Maxillary Sinusitis.—In chronic maxillary sinusitis the pain may be not only in the region of the antrum and reflected along the terminal nerve filaments of the second division of the fifth nerve, but also in the frontal region, even if the frontal sinus is absolutely normal or absent. To explain this phenomenon is not easy. As a matter of fact, involvement of any of the nasal sinuses may cause frontal pain. The probable reason for this occurrence is the fact that an irritation in any branch of the sensory distribution of the fifth cranial nerve may be the exciting cause of reflex pain in any other branch of the same nerve. As the frontal nerve is clinically known to be one of the most sensitive nerves of the body, an irritation in any part of the fifth nerve is easily reflected to it. Pain of this character is called by some referred pain. It is well, however, to call it reflex or reflected pain and to limit the term referred pain to autonomic pain.

Acute Frontal Sinusitis.—In acute frontal sinusitis the pain is either (a) constant with a sense of fullness and tension over the frontal region (periosteal pain) or (b) neuralgic in character, coming on in paroxysms, over the frontal region and radiating to the anterior temporal or parietal region. It

is due to disease or irritation of the terminal filaments of the nasociliary nerve of the first division.

Chronic Frontal Sinusitis.—In chronic frontal sinusitis the pain is usually not so severe and not so constant as in the acute form.

Acute Anterior Ethmoiditis.—In acute ethmoiditis the pain is either constant over the bridge of the nose or projected in paroxysms over the frontal and vertical region. It is due to involvement of the anterior ethmoid nerve of the nasociliary of the first division.

Chronic Anterior Ethmoiditis.—In chronic ethmoiditis the pain is of the same character but less marked. It may be absent.

Acute Posterior Ethmoiditis and Sphenoiditis.—In acute posterior ethmoiditis and sphenoiditis the pain may be (a) frontal, constant, due probably to disease of the sphenothmoid branch of the nasociliary of the first division, or paroxysmal, due to irritation of the same branch. It may, however, be reflex in origin; (b) retroorbital, more or less constant, due probably to involvement of the orbital branches of the second division, which supply the periosteum of the sphenoidal and posterior ethmoid cells as well as the periosteum of the orbit; (c) occipital, due probably to involvement of the afferent autonomic fibers.

Sphenoid sinusitis may cause neuralgic pains along any of three divisions of the fifth cranial nerve. This, according to Sluder,¹⁰ is due to the close relationship of the sphenoid sinus to the Gasserian ganglion. This would also explain the appearance of herpes during an attack of sphenoid sinusitis.

Chronic Posterior Ethmoiditis and Sphenoiditis.—In chronic posterior ethmoiditis and sphenoiditis the pain is similar in character, but less constant than in the acute forms of the disease. There are patients, however, with chronic ethmoiditis and sphenoiditis, who suffer the most agonizing pains imaginable.

7. Malignant Tumors of the Nose.—In malignant tumors of the nose pain is a prominent symptom. The tumor may cause pressure on the nerve endings or nerve fibers, and the site of the tumor will to a certain extent determine the location of the pain.

Pain in these cases may also be due to blood and lymph stasis. The tumor compresses the vessels of the nasal mucous membrane, which are in communication with the veins of the dura and the superior longitudinal sinus. The blood and the lymph stasis of the nose, causing a stasis in the dura mater of the brain, will produce pain.

8. Relation Between the Nasal Sinuses and the Meninges of the Brain.—It is well known that the nasal sinuses are anatomically closely related to the frontal cerebral lobes and the base of the brain. By means of the vascular and the lymph systems a communication between the nasal cavity and the meninges is brought about. The only meningeal membrane that is sensitive to pain is the dura mater. It is supplied in its anterior and middle portion by sensory terminal filaments of the fifth cranial nerve, and in its posterior portion by a branch from the sensory ganglion of the root of the vagus nerve.

Toxic absorption from the nasal sinuses seem to cause pain, which is not confined to the area of a particular nerve but is diffuse. It is most probably caused by irritation of the meningeal branches of the fifth and tenth cranial nerves and is usually accompanied by mental depression, inability to concentrate and loss of memory.

TRIFACIAL NEURALGIA.

In the preceding paragraphs pains due to nasal disease affecting (a) the nerve endings, (b) the nerve trunks, and possibly (c) the Gasserian ganglion were considered. They were described as constant and localized, or paroxysmal and neuralgic. The paroxysmal pain is an example of what is usually referred to as symptomatic neuralgia. It has absolutely no relation to what is called trifacial neuralgia or tic douloureux. In these cases the source of the pain is not to be found in the tissues of the organ from which it is transmitted to the sensorium through the sensory nerve (trifacial or fifth) supplying the tissues, but rather that it is due to some action on the sensory trunk itself.

The etiology of this terrible disease is obscure. Histologic investigations do not enlighten us fully. There is probably a biochemical change in the nerve, of whose nature we know

nothing. It is, therefore, necessary to keep in mind that a diagnosis of trifacial neuralgia is a diagnosis by exclusion.

REPORT OF A CASE.

A large number of cases could be cited that have been studied by me from the standpoint of the presenting symptom—pain—but space does not permit. I want, however, to report very briefly a type of case that we meet so frequently in practice.

A neurotic woman, 30 years of age, married, Hebrew, well nourished, came to the Manhattan Eye, Ear and Throat Hospital in December, 1921, complaining for two years of constant and severe pain over the frontal, vertical and occipital regions. She had been in this country only two years. She came from Hungary, where she suffered greatly during the world war. The pain she complained of was present only during the waking hours, never at night when sleeping. Stroking of the hair with a comb was very painful. She was examined by several physicians, who apparently did not find any organic trouble, and she was referred to the hospital for examination of the nasal sinuses. Anterior rhinoscopy showed a high deviation of the nasal septum, with pressure on the middle turbinates, and there was some mucopus between the latter and the outer nasal wall. Posterior rhinoscopy, pharyngeal and laryngeal examinations were negative. X-ray report by Dr. Law, December 31, 1921: "Left frontal shows mild involvement. Right frontal shows moderate involvement. Ethmoids show moderate involvement with considerable absorption of the posterior septa. Both antra show mild involvement. Sphenoids large and clear."

To give the patient the benefit of the doubt, a submucous resection of the nasal septum was done. The anterior tips of both middle turbinates were removed. The Loré suction irrigation apparatus was then used for six months. The pain has not been relieved. It is still present only during the waking hours and never interferes with sleep. The patient looks well and is gaining weight. She weeps and cries on the slightest provocation. Nasal examination at present is absolutely negative. Another X-ray examination was made on August 22, 1922, and Dr. Law reports all sinuses practically clear.

This demonstrates the efficacy of the nasal treatment, which cleared up the sinuses but not the pain.

Comment: This patient undoubtedly had a chronic sinusitis but her pains were not caused by it. Whenever a healthy looking and neurotic patient complains of constant pain, day by day, which does not interfere with sleep, its most probable origin is a neurosis. Before doing any operative work on the nose it should be remembered that psychic pain may on superficial examination simulate that due to nasal sinusitis, especially if certain signs of nasal disease or anatomic variations are present.

PHARYNX AND LARYNX.

It is impossible in a communication of this character to discuss all diseases of the pharynx and larynx that may cause pain. Only some phases of the subject which are of interest to the laryngologist will be considered.

HERPES OF THE PHARYNX.

Herpes of the pharynx is caused by an inflammatory condition of the sensory ganglion of the ninth nerve. It is usually not recognized. There is severe pain on swallowing, and a sticking pain in the throat even without swallowing. The pain may be so severe that patients will refuse food. Otalgia is also present. On examination we find vesicles surrounded by a red areola on the base of the tongue, on the palate or on the pharyngeal wall.

TUMORS OF THE NASOPHARYNX.

Malignant tumors of the nasopharynx may involve the Gasserian ganglion. It is important to remember that these tumors may not cause subjective symptoms until the skull is penetrated. Sheldon¹¹ of the Mayo clinic has observed sixty-nine tumors of the nasopharynx, nine of which have been attended with intracranial extensions. Of these nine tumors, four have exhibited the Gasserian ganglion syndrome. Pain is usually the initial symptom and the one for which relief is sought.

LARYNX.

Pain is present in all acute pus infections of the larynx. Its intensity depends on the location of the lesion. In infections

of the epiglottis and the arytenoepiglottidean folds the pain is most severe, because this area has a very rich sensory distribution, and also there is here more irritation on swallowing. In herpes of this area the pain is so severe that it is impossible to take any nourishment. It is due to an inflammatory process of the sensory ganglion of the vagus nerve. It may, therefore, be accompanied by other vagal symptoms, like frequent vomiting, slow and irregular pulse and otalgia.

In chronic diseases of the larynx, like tubercular ulcers and extralaryngeal carcinoma, pain is a prominent symptom. In tuberculosis of the larynx the pain may be so severe that it is justifiable to inject the superior laryngeal nerve with alcohol at its place of entrance through the thyrohyoid membrane. In carcinoma of the larynx the pain depends upon the location of the tumor. Extralaryngeal tumors give rise very early to pain on swallowing, which may be the first presenting symptom. We should not forget, however, that lesions in this area may cause reflex otalgia. I know of a patient who came to the Lenox Hill Dispensary complaining only of pain in the right ear. Examination of the ear was negative, but an involvement of the right half of the larynx was found. Microscopic examination confirmed the diagnosis of extralaryngeal carcinoma of the larynx.

In *tabes dorsalis* laryngeal pain, of a burning and lightning nature, is a prominent symptom in the so-called laryngeal crisis.

THYROIDITIS.

In thyroiditis the thyroid gland, as a whole or in part, becomes swollen and tender. There is pain on swallowing. The pain may radiate to the region back of the ear. Two such cases have been seen by me at the Manhattan Eye, Ear and Throat Hospital. They both complained of pain on swallowing and of a choking sensation. The thyroiditis in both cases was preceded by an acute tonsillitis. When seen at the hospital no acute inflammatory condition was evident in the throat.

ANGINA PECTORIS.

Occasionally pain in the throat and teeth are the only manifestations of an angina pectoris. The diagnosis is made on

the evidence of arteriosclerosis, pain on pressure of the carotid arteries and typical anginoid attacks in the interval.

GLOBUS HYSTERICUS.

Globus hystericus is a painful constriction in the throat, due to a hyperesthesia of the esophageal and pharyngeal mucous membrane, causing a contraction of the musculature of the esophagus and pharynx. The diagnosis of this condition is easy if the patient has the classical feeling of a globe rising from the cardiac end of the stomach behind the sternum to the throat, where the globe gets "stuck," and if other hysterical symptoms are present. But when the patient complains of a painful feeling of constriction alone in the throat we should be careful not to call it a globus hystericus. We are more liable to make the mistake if the patient is a more or less nervous woman. We should remember that it may be the only symptom of an angina pectoris. Another cause of a feeling of a constriction in the throat may be a heart insufficiency, which produces a congestion and a sensation as if the blood rushes to the throat. Hyperthyroidism may produce such a feeling with a sensation of choking. It is also very frequently caused by flatulence, after a large meal, especially where there is a slight cardiac insufficiency.

Direct irritation of the pharyngeal branches of the vagus nerve which supply the muscles of the pharynx may produce a contraction of these muscles and a painful feeling of constriction. This irritation may be brought about by mediastinal disease, by intrathoracic goiter, by aneurysm of aorta, etc.

An example of a possible hyperirritability of the pharyngeal branches of the vagus nerve without discoverable cause was recently seen at the Manhattan Eye, Ear and Throat Hospital. A perfectly healthy young man came to the hospital complaining of a choking sensation and a painful feeling of constriction in the throat. Examination of the throat was absolutely negative. Atropin sulphate, which has a paralyzing effect on the vagus nerve, was given in gr. 1-100 doses, twice a day. After taking a few doses of the atropin the symptoms were gone. Occasional treatment with the same drug prevents the re-appearance of the symptoms.

HEADACHE (DIFFUSED PAINS).

The limitations of this paper forbid a complete discussion of headache. A few points, however, of interest to the laryngologist will be emphasized. We ought to be able to distinguish between pains of the head and headache. Pain, being due to irritation of the nerve endings or nerve fibers, will be more or less localized. Headache, on the other hand, being due to toxic absorption from the diseased tissues and to an involvement of the dura mater is diffuse. Pain, therefore, is a local symptom, and headache a focal symptom. The focus of infection may be in head, neck or in some other part of the body.

It is known that there is a more or less intimate connection between the vagus centers and the fifth cranial nerve. Thus Head¹² has described certain cephalic zones which he considers particularly involved and painful in the presence of disorders of the thoracic and abdominal viscera, which are supplied by the vagus nerve. These zones are at present of not much value to us. It should be remembered, however, that a good dose of castor oil will sometimes relieve a headache that is supposed to be due to nasal disease.

INDURATIVE OR RHEUMATIC HEADACHE.

Another type of focal headache is the one usually referred to as indurative or rheumatic headache. It is characterized by fibrous deposits or nodes at the insertions of the scalp and neck muscles. It occurs mostly in the occipital region and is worse on motion and in damp and windy weather. A characteristic symptom of these patients is the dizziness they complain of, especially on bending down. It is most probably due to a disturbance of the sensory nerves of the involved muscles. Normally these nerves of deep sensibility transmit to the central nervous system sensations of position and movement of the head and neck. In disease of the muscles implicating these nerves the central nervous system receives false impressions on movement of the head and neck which leads to dizziness.

MIGRAINE.

Migraine is an hereditary and obscure type of headache. It is a periodic neurosis in which there is a nervous discharge

affecting not only the fifth but also other cranial nerves as well as the autonomic nervous system. It is a general disease in which the headache is only one symptom. There are many theories to explain its causation. The one most accepted by neurologists is that it is due to a vasomotor disturbance, which causes an intracranial vasoconstriction and an anemia of the brain. If we accept this theory, the question still remains: "How is the pain produced?" That a portion of the brain, for instance the sensory nucleus of the fifth nerve, can be irritated by an anemia does not appear rational. According to Lewandowsky,¹³ the pain is possibly due to the contraction of the walls of the blood vessels of the brain. Nasal disturbances have also been thought to be causative factors.

HEADACHE IN ENCEPHALITIS.

It should be remembered that violent headache may not only accompany but also be one of the sequelæ of epidemic encephalitis. It may occur months after the process has subsided. If this is not kept in mind mistakes in the diagnosis are easily made.¹⁴

PITUITARY HEADACHE.

Pituitary headache is due to a disproportion between the sella turcica and the pituitary gland, producing pressure on the sensory nerves of the dura mater. The pain is usually bitemporal or deep in the forehead behind the eyes. It is relieved by the administration of the whole pituitary gland in about one grain doses, three times a day. The following case may serve as an example:

A man, 32 years of age, bookkeeper, came to the Lenox Hill Dispensary, on August 28, 1922, complaining of pain deep in the forehead behind the eyes. He suffered this pain for the last four years. It always appears after exertion, like going upstairs, or after any kind of exercise. His eyes were examined by several ophthalmologists, who prescribed eyeglasses. The pain, however, was not relieved. He used to take a great deal of aspirin, which weakened his heart but had very little effect on the pain. Before he came to the dispensary he was treated for "heart trouble" in one of the leading hospitals of New York. There he was advised to have his nose examined. It was thought that his pain was due to a nasal sinusitis.

Examination showed that the nose and throat were absolutely negative. X-ray report, August 30, 1922, Lenox Hill Hospital: The frontals, ethmoids and antra do not reveal any pathology. The sphenoid sinuses show cloudiness at their posterior part. The sella is small, the posterior and anterior clinoids appear to meet. The dorsum sellæ is irregular and thinned out.

The patient was given pituitary extract, grain 1, three times a day. After a week's treatment the pain practically disappeared and has not returned up to the present time (December, 1922).

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161 EAST 79TH ST.

LIII.

RECENT NASAL FRACTURES.

BY IRA FRANK, M. D.,

CHICAGO.

It is to be regretted that current literature so rarely brings to the mind of the student and general practitioner the pressing need of proper care and attention to fractures of the nose. It is the universal opinion of those experienced in this work that there are few cases of fractures in any location on the skeleton that require more constant care and vigilance than fractures of the ossa nasi; and it may be added that there is no facial organ more sensitive to deformity in its relation to disfigurement of the face.

Far more was written on this subject in the uncertain past than appears in the periodicals of the present day. The older recommendations consisted principally of enthusiastic descriptions of forceps, splints and complicated mechanical devices calculated to hold the set nose securely in place until union resulted, apparatus which at the present time is no longer heard of, or which was even disavowed later by the authors themselves. Very few of the procedures in the long history of the subject survive, not through wrong effort or lack of ingenious design, but rather because the various mechanisms submitted were built to meet individual case requirements, hypothetical cases or artificial lesions produced on the cadaver. The error was for the most part due to an effort to treat all fractures of the nose by a single method. Considering the great varieties of nasal fractures, due to varying degrees of force applied in varying directions by objects of all shapes and sizes at different levels of the nasal bridge, it is readily conceivable that the number of lesions to be produced is infinite, and it is evident that no single method or immobilizing device can be applicable to such a multiform lesion. The older practitioners would seem to have failed to realize that probably no man sees two cases of fracture of the nose exactly alike.

II.

Injuries of the nose may be divided into two primary classes—injuries without loss of tissue, and trauma with destruction of bone, cartilage and soft structures; and as a result of the varying degree of force in varying directions the resulting injury ranges from minor dislocations of the columnar cartilages to complete crushing injuries of the nasal arch and septum.

The supporting structures of the nose, whether osseous or cartilaginous, are subject to three classes of trauma—i. e., dislocation, infracture and complete fracture, and all cases of injury will fall under one or more of these classes. The involvement of the septum is, however, usually limited to dislocation and fracture.

Injury to the septum in a large proportion of cases is of course due to its anatomic position as a rigid central support for the bridge of the nose, and from its exposed position the quadrilateral cartilage suffers more frequently, though to no greater degree than the osseous portion. Zuckerkandl, whose figures are widely quoted in this connection, found that in 80 per cent of the skulls examined the bony septum (perpendicular plate of the ethmoid) extended as far down as the middle of the long axis of the nasal bones or even lower, and in only 3 per cent did not reach to the upper margin of the ossa nasi.

Practically all fractures of the nose are of the compound type, and this point must be kept in mind during the entire course of treatment, even though, as occasionally happens, there is little or no loss of blood from the nares to indicate laceration of the mucous membrane. It is common practice, due doubtless to the infrequency of complications, to disregard this factor, which is unsurgical to say the least, and which has led to serious and even fatal complications. There is not, it is true, the same degree of serious significance attached to these compound fractures as attends those of other parts of the body, immunity being granted by several probable factors. The injured bone is ordinarily thin and shell like, consequently the exposed broken edges are very thin and are readily sealed by the coagulation of blood coming from the minute sinuses

and capillaries of the injured and exposed surface. The marked vascularity of the supporting and lining structures of the nose doubtlessly confers a local immunity to infection during exposures of the tissues, and finally we may consider that because bacteriologic examinations of the normal mucous membrane show attenuated strains of bacteria present in a locus theoretically believed to hold a special immunity to those germs normally present, danger is remote except in the presence of complicating infections, such as rhinitis and sinus disease.

III.

The size, form and general appearance of the injured feature may be greatly altered by swelling, ecchymosis and the bruising of surrounding soft tissue, the reaction being so great that the detection of fracture and dislocation is extremely difficult and uncertain. When the swelling has at least partially subsided, or in those cases where the reaction to injury of the soft parts has been less severe, it is possible to obtain an exact idea of the extent and degree of injury by external and intranasal examination. The deformity presented by simple inspection often reveals the nature of the injury without palpation or radiograph, but the importance of careful intranasal inspection cannot be overemphasized. The septum may be dislocated and so much displaced laterally as to considerably narrow or even completely occlude one nostril without altering the external contour of the nose.

Dislocation.—The most frequent cartilage dislocation is quite naturally a separation of the quadrilateral cartilage at its junction with the perpendicular plate and with the vomer. This results usually in an apparent and actual depression of the flexible portion of the external bridge, with the tip of the nose frequently directed to the side of the dislocation. If the septum, as a result of direct violence, is dislocated backwards the resulting deformity is fairly typical; a deep depression immediately beyond the lower extremities of the nasal bones and a slight but unnatural tilting up of the tip of the nose. Both of these types of dislocation are accompanied by partial or complete separation of the cartilage from the nasal process of the superior maxilla, complete separation being responsible

for the frequently observed widening of the columella due to encroachment of the anterior angle of the cartilage into the nostril.

Infractures.—Infractures of the cartilaginous or osseous structures of the nose offer perhaps the most annoying difficulties of diagnosis, and it is in the majority of these cases that unsatisfactory end results are obtained. With crepitus absent and no help lent by the Roentgen ray, and with all acute swelling apparently subsided, the perichondritis or periostitis which may persist for weeks disguises a cosmetic irregularity in a manner likely to indicate a satisfactory prognosis which may later be modified by cruel fact. Tactile examination must indeed be highly specialized to determine depressions of infractures hidden by firm indurated periostitic swelling.

Fractures.—The concept regarding the osseous structure of the nose as an architecturally supported arch is apparent in the writings of earlier surgeons and is sometimes met with in the present day. That the nasal projection is a poor substitute for an arch from the engineering standpoint is readily conceded by those who have frequently attempted its reconstruction.

The extreme rarity of nasal fractures compared to injuries of other bones of the skeleton must not be ascribed to any special resistance of the so-called arch itself, but rather to the infrequency with which the organ is subject to injury. It is well to remember in considering this point that the same remarkable automatic protection of the eyes by instinct—i. e., the jerking of the head or the raising of the arms to ward off impending blows, does more to protect the nasal bridge than does the structure of the bridge itself. It is a fact borne out by a study of comparative anatomy that the skulls of mammals of the lower orders show a far greater support of the nasal arch and bridge (up to and including the negroid skull) than is present in the Caucasian race.

Statistics on fractures in general from the central European medical centers show nasal fractures to have a frequency of from 1 to 1.6 per cent. According to the few available hospital reports in this country, these figures seem high, due either to the fewer number of adherents to dangerous sports or to the

fact that many of these cases are considered ambulatory and are privately treated in physicians' offices.

In practically all traumatizations of the nose with external direct force to the osseous bridge there is a tendency of the nasal bones to separate from each other, as there is practically no obliteration of the central suture in the white race. Carter states that bony union is frequently seen in the negro and Mongolian races, and that there is constant osseous union in apes. This tendency to separation is responsible for the great frequency of dislocation of the nasal bones. It is doubtlessly true that the nasal bones are more frequently dislocated from the frontal processes of the superior maxillæ or separated from each other than actually fractured.

The most common injury then involves the separation of the nasal bones and their fracture or both. This condition presents marked deformity depending on the force applied. There is an overriding of the nasal bones on the nasal processes, a flattened deformity of the bridge resulting. Marked flattening is accompanied by backward dislocation of both nasal bones and septum, and may be of sufficient degree to produce an undue prominence of the nasal processes of the superior maxillæ, causing an apparent widening of the distance between the eyes.

With a forcible blow striking the nose from the side, one or both nasal bones may be fractured and displaced from their beds and the organ is pushed to one side. In blows from the same direction but applied with lesser force only the nasal bone of that side is broken with a depressed deformity. Laterally applied blows to the bridge of the nose in children may produce a depressed deformity due to fracture on the same side and a greenstick or infraction of the opposite nasal bone.

The chief lines of fracture traverse the lower half of the nasal bones in a more or less oblique direction; the ossa nasi are thinnest at the lower extremity, requiring only a light blow properly applied to cause a separation of continuity. The fracture is frequently seen to traverse the bone at a point corresponding to the nasal frontal spine. Occasionally the break may be seen to run longitudinally following the grain of the bone, especially when the force has been applied with a small object.

Fractures of the septum may occur independently of fractures of the nasal bridge, and, as Jaravay pointed out years ago, innumerable fractures of the cartilaginous septum occur without any injury to bone, and with so little discomfort or reaction the lesion may remain unknown to the possessor.

The most usual location of fractures of the septum is in the posterior two-thirds of the cartilaginous portion and the anterior half of the osseous portion, the latter usually in the perpendicular plate, the vomer being protected by virtue of its deep position.

IV.

Hippocrates thought that union of the fragments in nose fractures was generally complete in six days. Boyer, Maligne and other early writers believed that these fractures healed with great rapidity. Molinie stated that eight days as a minimum is necessary for fixation. It is rather conceded by contemporary writers that fixed union does not occur till late in the second week or well into the third.

Correction of old septal deformities disclose a difference in the method of natural repair of fractures of bone and cartilage. Osseous repair or union is made by means of hyperplastic callus and new bone formation. Repair of cartilaginous fractures is by simple connective tissue callus.

V.

The complications resulting from fractures of the nose are many and varied. Probably the most ordinary complication in practice is a septal hematoma, which may lead to abscess formation. Low lateral injury may cause laceration of the lacrimal duct. The line of fracture may extend upwards into the frontal bone and frontal sinus, causing an emphysema when attempt is made to blow the nose. The same type of injury in greater degree may cause the line of fracture to reach the anterior fossa with accompanying symptoms of cerebral concussion and other intracranial injuries. Rogers has reported three cases of fracture of the cribriform plate and death from meningitis. Severe crushing comminuted fractures have involved the maxillary sinus and even anterior ethmoid cells. A late complication is chronic suppurative periostitis or perichondritis, recognized by fetid odor, purulent discharge and

pain. In these cases the condition persists, and the nose remains sensitive until necrotic bone or cartilage is discharged by nature or removed surgically. Of the rarer local complications cavernous sinus thrombosis and ophthalmoplegia (Dowling) may be mentioned.

VI.

Treatment of nasal fractures is best considered in two divisions: (a) reduction and (b) retention. The question of when to undertake these measures is still open to discussion. Garel, Harold, Foster, Lee Cohen and others insist on an immediate institution of treatment, while many operators feel that a more thorough diagnosis and a more exact approximation of the separated parts can be made if the circulatory reaction following the blow has subsided. I have personally set no hard and fast rule to cover the question and allow myself to be governed by the individual case. A fair proportion of these injuries show surprisingly little swelling and edema, and in many the swelling from the circulatory disturbance is soft enough to permit thorough tactile examination. When the reaction is severe enough to prevent a complete study of the supporting structures I allow a period extending up to ninety-six hours in which to allay the reaction by the use of ice packs, rest in bed and elevation of the head. At the end of this period I feel certain enough of the condition of the nose to perform reduction.

(a)

Almost every known instrument with proper slenderness and rigidity has been used and recommended for the intranasal reduction of fractures. Septal periosteotomes, female silver catheters, Volkmann's curette, the Tripier dilator, artery snaps, the Martin forceps come to mind as recommended by various authors. A number of special forceps for reducing the septum have been devised, of which the most popular is doubtless the Adams instrument, especially as modified by Carter.

The rules governing the reduction of nasal fractures vary almost as greatly as do the types of injuries. Simple reduction by traction with subsequent natural retention, as in fractures of the long bones, is possible in so few of the cases that

the percentage is practically negligible. Reduction is usually a matter of reconstruction of the superstructure of the nose with immediate support, which must be maintained until rigidity is established. Dr. Lee Cohen believes that this can be accomplished with best end results by thoroughly mobilizing the nose with the Adams-Carter forceps, eliminating all resistance of the injured or uninjured supporting structures, so that when placed in a splint the nose may be molded into proper form.

(b)

For intranasal retention of the reduced fracture a large variety of methods have been used. From the earliest days of lint packing, splints of various design and material have been conceived. Rubber tubing, cups of rubber and gutta percha, hollow tubular splints of rubber and metal have been hailed with enthusiasm and then forgotten. Probably the gauze pack and the Simpson sponge are the only examples of this type of retention apparatus which we use today.

Of the mechanical devices few have been of practical value. Probably the most successful of these is the apparatus conceived by Martin and Martin; a four pad mechanism arranged winglike, which is introduced into the nostril closed. A plate resting on the floor of each nostril acts as an anchor, and the turning of a key outside of the nose opens the wings of each side simultaneously so that one pad exerts pressure against the lateral wall of the nose while another applies itself against the septum wall. Molliere, Chandeloux and more recently Nelaton, Ombredanne and others, find that the apparatus is seldom tolerated by the nasal mucous membrane, though the Martins maintain that the nose can sustain the apparatus for long periods of time with proper regulation of tension. Variations of this apparatus are used by the Martins, Sargnon and others for the correction of recent fractures and deformities.

Molinie devised an internal expanding splint which is now recommended chiefly for the treatment of old fractures. The opened splint gives no lateral pressure, but being triangular in form and resting on the floor of the nose with its base, it exerts tension along the entire line of the bridge close to the median line.

The external splints are as varied and almost as complicated in some instances as the internal splints. Molliere introduced his external plaster method in 1888. Prior to this time other external apparatus had been devised, such as the lead gutter of Malgaigne, molds made of rubber (Hamilton), papier mache (Dzondi), dachylum (Weber), collodium (Dumreicher) and dental plate composition (Garel).

Warbasse recommends as recently as 1918 a U-shaped rigid appliance attached to a headband descending from the forehead to enclose the nose, from the side of which padded thumbscrews are applied to exert pressure at desired points. This apparatus is further improved by a flat metallic tongue attached to the bottom of the U so that it lies over the upper lip. This permits varying degrees of counter pressure by applying a bandage around the head above the pinnæ and beneath the nostrils.

In this country the apparatus of Carter, devised as an external support to the nasal bridge, was at one time popular. This consisted of a rigid adjustable arch set over the nose, its base resting on the frontal processes of the superior maxillæ. Splints of gutta percha were inserted and adjusted in the nostrils with thread and needles attached, which were passed through the tissues of the nose and secured externally to the rigid arch. Frequent loosening of the tension and bathing of the parts are said to have prevented necrosis.

A modification of this splint was suggested by Grant in 1903. This writer omitted the use of the internal splints and passed silver wire through the depressed deformity, attaching it to a steel arch straddling the bridge, the arch resting on rubber cheek plates retained by means of tapes passing around the head.

Other external mechanical splints have met with more success. When used in selected cases the device of Adams has been very satisfactory. It consists of two lateral pads fastened by rigid swivels to a firm head band and adjusted against the side of the nose by a thumbscrew. This is similar to the adjustable pads of Joseph of Berlin. Gamgee's apparatus presses on the side of the nose by means of a screw and spring, with lateral movement of the apparatus prevented by means of a spectacle frame hooked to the ears. Cobb later devised a

similar appliance, held in place by a stiff metal head band fitted carefully to the circumference of the head.

The ingenious device of Von Eck deserves mention. It consists of a rigid metal saddle held over the bridge of the nose by buckled tapes around the head. Through this saddle are inserted six adjustable thumbscrews, set in various positions, which press on a flexible metal plate in direct contact with the nose. By lowering or raising the several screws the inner plate, which is padded, can be made to conform to the requirements of many individual cases.

Most rhinologists have a collection of many of these devices locked away in cabinets reserved for tonsillotomes. It is wrong to condemn them all as useless, for many of them are applicable to selected cases; the principal objection is the extreme rarity in which a given apparatus may be properly applied.

The sheet copper splint, which Cohen attributes to Roe, but which practically is identical with the zinc splint of Daly, is proving to be the best retention apparatus we have at our command. To properly construct the splint, which is made to fit each individual case, a paper pattern saddle is cut to fit the injured nose, and the resulting truncated triangle is used to cut a similar shape from a sheet of 24 gauge sheet copper with tinner's shears. The edges of the copper are smoothed with a file and the entire surface and edges are covered with a layer of adhesive plaster and an inner lining of soft thin cotton.

TECHNIC.

Dr. Cohen believes, and I am inclined to believe that most of us with experience in this work agree with him, that it is most essential in the reduction of all fractures to first thoroughly mobilize the entire bony framework of the nose, so that when placed in the splint it may be molded into proper form. Unless this is done reduction cannot be satisfactorily accomplished, and even if the reduction seems to be successful there is always a tendency of the nose to assume a faulty position, which grows more manifest as the swelling disappears.

To produce mobilization (I am quoting Lee Cohen at length) the nasal bone is grasped with the Carter-Adams forceps, one blade of which is introduced into the vestibule of the nose and the other (covered with rubber tubing to prevent injury to the skin) is placed on the outside. The bone is then twisted with sufficient force to assure complete mobilization, and the same process is repeated on the other side.

Following this the same forceps are reintroduced, both blades bare, into the nostrils, one blade on each side of the septum, so that, by a lifting motion or twisting, the nasal bones can be brought forward and upward into their proper relation with the nasal process of the maxillary bone below and the frontal bone above. This manipulation serves at the same time to bring the septum of the nose into proper alignment.

To retain the desired position the nasal chambers are lightly packed with plain pulled gauze in continuous strips one-half inch wide, care being taken to elevate with firm but gentle pressure those portions showing a tendency to depression, in certain marked tendencies an overcorrection being advisable. The light resting of the thumb and index finger externally with one hand during the packing serves to guide the operator's judgment.

The copper splint just described is then placed on the nose and is held in place by an inch wide strip of adhesive tape extending across the nose and face from beneath one ear to the other. A small strip of adhesive plaster extending from the forehead down to the lower end of the splint, reinforced by two cross strips on the forehead, prevents any tendency of the splint to slide downward and exert undue pressure on the tip of the nose.

It is important to remember that great care must be taken to insure an equal tension to the cheek strips holding the splint in place, as an uneven pull will easily cause the axis of the nose to get out of alignment.

The first dressing is left for four days, and then changed every third day. If, however, there appears to have been any displacement of internal packing or external splint during daily observation, it must of course be redressed at once. The

packing may be discontinued after ten to fourteen days. The splint should be used from three to four weeks.

In conclusion let three elementary principles of general surgery be recalled in the treatment of these cases: First, asepsis, especially in the compound fractures; second, suture of lacerated skin with a view of minimizing scar formation, and third, the use of tetanus antitoxin when indicated by the nature of the injury.

THE OTOLOGIST IN THE FIELD OF SOCIOLOGY
(THE ALLIANCE OF MEDICAL AND SOCIAL
SCIENCES IN THE PREVENTION AND PAL-
LIATION OF ACQUIRED DEAFNESS).*

BY WENDELL C. PHILLIPS, M. D.,
NEW YORK.

Luke, the beloved physician, records an incident in the life of the Nazarene when a certain lawyer sought a reply to the question "Who is my neighbor?" His reply was as follows: "A certain man went down from Jerusalem to Jericho, and fell among thieves, which stripped him of his raiment, and wounded him, and departed, leaving him half dead. And by chance there came down a certain priest that way; and when he saw him, he passed by on the other side. And likewise a Levite, when he was at the place, came and looked on him, and passed by on the other side. But a certain Samaritan, as he journeyed, came where he was; and when he saw him, he had compassion on him. And went to him, and bound up his wounds, pouring in oil and wine, and set him on his own beast, and brought him to an inn, and took care of him. And on the morrow, when he departed, he took out two pence, and gave them to the host, and said unto him, Take care of him; and whatsoever thou spendest more, when I come again, I will repay thee. Which now of these three, thinkest thou, was neighbor unto him that fell among the thieves?" The lawyer answered: "He that shewed mercy on him." Then said the Nazarene: "Go, and do thou likewise."

To the physician disciple this parable must have made a strong appeal, for to the physician, not only on account of the nature of his avocation and the Hippocratic oath to which he has subscribed, but because of the human element in his experience and training, responsibility as neighbor to his pa-

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tient becomes imperative. The peculiar psychology surrounding his deafened patient vastly enhances this responsibility. Every handicap in human experience has its psychologic bearings; every calamity in life its woes; but none except obvious fatal diseases provokes more absolute helplessness and depression than defective hearing.

In the presence of this audience of otologists I need not attempt to enlarge upon this phase of the hearing problem. I know of no more trying experience than to be called upon by a deafened patient to give a final opinion, when no hope of permanent improvement may be held out. We older otologists frequently are obliged to face this ordeal. In the functioning of this experience the prevention of deafness plays no part, for in every instance the days of prevention have passed. Likewise in the majority of cases no well founded hope for improvement can conscientiously be vouchsafed. We must face the bald facts surrounding his deafened condition, not only in the patient's interest, but it must be in a manner that will preserve our own integrity and honesty of purpose. At this very point arises the otologist's opportunity to become his patient's neighbor in the highest sense, and it is here that he may profitably enter the field of sociology, at least so far as sociology as a science may pertain to the welfare of his deafened patient. It is one thing for the otologist, after having conducted all the hearing tests, to conscientiously tell a deafened person, even in the kindest way, that his deafness must remain permanent. It is quite another attainment to go to the necessary length of demonstrating to him how to overcome his handicap. The first is an ordinary professional business transaction—the second requires time, self-sacrifice and toil beyond the realm of financial remuneration. The otologist who contents himself with the rendering of his scientific opinion only, must be classed with the priest or the Levite who came and looked on and passed by on the other side. The citation of a few experiences will serve to illustrate:

A boy of fourteen summers whose mother stated to a social worker that she had formerly consulted a number of otologists, who all agreed that her son's deafness was incurable, but they failed to tell her what could be done to lessen the handicap. At last she consulted another otologist (not the writer), who

is deeply interested in organization work for the deafened and who is constantly in touch with its workers. He told her that her son must study lip reading without delay; that his vocation must be carefully selected and his whole trend of life guided in the direction of an avocation which he could fill with satisfaction to himself and to his friends. He was placed in a private school of lip reading. Later he entered a high school, taking a cooperative industrial course, and finally landed a remunerative job. His mother's gratitude overflowed her vocabulary—her problem was solved at last. "That—that—is a hero man" was her cry.

A young woman about twenty-two years of age consulted one of the most distinguished members of this society. His attitude was friendly, his examination thorough, every test complete and painstaking, leaving nothing unaccomplished that could add to the thoroughness or the value of his scientific opinion. Upon completion of the examination he told her plainly that her deafness was permanent and that no form of treatment held out any hope for permanent amelioration. She paid his fee and went home to spend the days of agony, hopelessness and despondency of those who must remain in the "silent land." She saw no ray of hope, either in the way of occupation, of social enjoyment or of education—the whole world was dark. Finally, some friend persuaded her to consult another otologist, who arrived at exactly the same hearing results, but who devoted the required time in an endeavor to open the door of hope. He explained to her that her defect was her peculiar handicap, that people all over the world with few exceptions were victims of one handicap or another. He explained the peculiar psychosis of deafness; how it grappled with one's optimism; he related incidents where people far worse than she had come into lives of comparative happiness and usefulness; he urged her in the strongest terms to rebuild her life on the basis of her handicap by making a careful study of lip reading, by using artificial aids for hearing, by associating herself with other deafened people in the field of social service work and by preparing for remunerative employment. She was introduced to the social service workers in the New York League for the Hard of Hearing and entered actively into its work. She was told that while no direct

benefit might come from constant treatment, there were times while suffering from severe colds that a moderate amount of nasal and aural treatment would be beneficial. It was impressed upon her that she could remain under the advice and counsel of a competent otologist who would be primarily her guide and counselor regarding all things pertaining to the hearing function. She responded to these suggestions with eagerness. This young woman has completely adjusted herself to her handicap and is happy. She is following a useful and remunerative occupation. She is becoming a competent lip reader and, strange to say, actually thinks she hears better than formerly.

One of my own experiences was a woman of about thirty-three years of age from a distant city, who had gradually from her twentieth year grown deaf from otosclerosis. I was called upon to say the final word and could not conscientiously evade the responsibility. She was despondent, rebellious, belligerent and refused to be comforted. She would not even consent to investigate the various artificial aids for hearing. I finally obtained her consent to go with me and confer with some people who are much deafer. I called at her hotel and took her to our League headquarters, where we spent an hour in a conference with our deafened workers, all of whom are happy and contented. We persuaded her that the wearing of a hearing device tended to make friends and to retain them, for the reason that one's friends are glad to talk into the hearing device, knowing that no special effort is required. She was shown through the various departments; educational, vocational, occupational, etc. She was urged to make a study of lip reading and to learn the things that would be of the greatest benefit in overcoming the handicap of deafness, and finally she succumbed to the influence.

Hundreds of cases of this kind might be cited through various organizations of the hard of hearing throughout the land. In another communication* I cited the case of a boy who had gradually grown deaf from the age of thirteen until twenty. When he graduated from high school he refused to enter college, stating as his reason that he did not like to

*Volta Review, November, 1921.

impart to his mother that he was afraid to go through the same deafened experience in college as in high school. It was at this time that I advised him to join our League for the hard of hearing and take advantage of its various departmental work, particularly lip reading. In a personal communication received from him he relates his League experiences. His letter says: "I have started one of the strangest transformations in my life. I have begun to look at the world in a different light. You advised me to join the lip reading class as soon as possible, and I attended one at the League rooms. I will never forget my first night of lip reading. I came full of hope, for I had been told that lip reading was a wonderful thing and expected to learn the entire art in one lesson. How my hopes were dashed to pieces. All around people were nodding their heads in the negative or affirmative as the case may be; at times they would burst out in laughter at something the teacher had said. I sat there all alone watching them and cursing myself—what mockery, what foolishness. I almost cried, but as time went on, my point of view gradually changed—I found out it wasn't so bad, after all. That a person who was deafened did not have to isolate himself from the world. At the present time I have accomplished a great deal in lip reading. I also come into contact with other activities of the club, especially its social life. I have taken part in three or four amateur dramatic performances. I joined the dancing class; in fact, my entire life has been changed by this experience. Sometimes I pinch myself to see if it is all real. Words cannot express the work that the young women of the League are performing. Their cheerfulness and happiness take one out of the deepest depths of gloom and misery." This young man is now in his senior year in a dental school and is accomplishing his work with very little difficulty.

The above remarks give emphasis to the fact that the otologist's responsibility does not end with the medical care of his deafened patient. He must carefully weigh every factor that will tend, first, to the elimination of his hearing defect, providing such relief is possible, but in the more serious cases he should assume the attitude of the Samaritan in the parable and become the neighbor, the teacher, adviser and guide of his

deafened patient. He should be prepared to educate the public, particularly along the lines of preventive deafness; to recommend the prophylaxis in individual cases, and he should encourage all organized efforts to ameliorate the unhappy condition of deafened people. In short, the otologist, together with the social worker in charge of the necessary activities for the deafened, must impart the necessary courage, information and hope to enable him to rebuild his life upon the new basis. "In the metamorphosis from the morbidity and shrinking self-pity to happiness and usefulness" more or less character rebuilding and readjustment become necessary.

SENSIBLE SUGGESTIONS FROM THE DEAFENED INDIVIDUAL'S
STANDPOINT.

Mr. John de Raismes Storey, in an article on the "Psychology of Deafened People,"* emphasizes the necessity of overcoming the obstacles within as well as those which are objective. Deafened himself, he refuses to be a typically deafened man and endeavors to be successfully deaf. To use his own words: "By that I mean he must strive to live a moral, wholesome life with an occupation suited to his capacity, with friends, hobbies, wide interests, etc., and not to let his affliction affect his character except beneficially." "I do not feel that the deafened man should accept a humble task if he is fitted by attainments for a better one." These are the sentiments of a deafened man who has achieved success in the legal profession and who is in active practice today.

At the third annual meeting of the American Federation of Organizations for the Hard of Hearing, held in Toledo, June, 1922, interesting papers were presented by deafened people representing a diversity of occupations. "The Deafened Man in Business Life" was discussed by Mr. Starling W. Childs of New York City, who is a member of a prominent brokerage house. When Dr. Phillips asked me to come here he gave me as a subject, "The Deafened Man in Business,"† and I took it. He meant me to discuss this from my own experience rather than to treat it along more theoretical

*Volta Review, May, 1920.

†Volta Review, Nov., 1922.

lines. I have done this, and you will understand that what I have to say today I have tested and found true by the best of tests—day by day experience. And by the same token, you will also understand that I am not dealing with the man who is totally deaf, but rather with the question of how people who suffer from various degrees of impaired hearing may be expected to get along in the world of affairs. Persons who are hard of hearing hold within themselves the key to their own destiny. If they allow themselves to be weak, discouraged and despondent they will for a time receive the sympathy they obviously seek, but if they persist the chances are that they will first be pitied, then endured, and then avoided. "Instead of weakly deploring our hard lot, we should strike a balance (as the business man would say), wherein our assets, as well as our liabilities are listed, and by steadily conserving the former and improving them we can be reasonably sure of some day showing a balance on the right side of the ledger." We are often bored by friends with discussions of their general ailments, and in a way they seem proud of them, but the deafened man—almost never. This probably arises from the fact that deafness is not an obvious defect, but the attitude is a wholly mistaken one and is sure to be a great obstacle to his success. Instead of concealment and vain subterfuge, his attitude should be one of complete and unembarrassed frankness. By frankly admitting his difficulty he will at once put those about him at their ease, and I can say with absolute assurance that he will almost invariably find his fellow men helpful and sympathetically cooperative. And do not forget that where real study of a subject or piece of business is required the deaf man has one very positive advantage. By shutting off his hearing apparatus he is at once free of all the disconcerting and disturbing noises which constantly assail the ears of his associates, and is thus enabled to concentrate absolutely upon the problem before him. In short, the deafened man will find by experience that his balance sheet has assets as well as liabilities. He then commends the employment of hearing devices and concludes with the statement that the deafened man is better equipped for some kinds of work, particularly in accounting and other business which require great concentration (In short, Mr. Childs has shown

his power of concentration by winning several matches of golf from the writer of this paper.)

"The Deafened Woman in Society" was a paper presented by Mrs. George L. McAlpin,* who stated that even if modesty causes one to shrink from relating personal experiences, for the cause of humanity and for the sake of service one would not remain silent. To sympathize, to help, to encourage, these must be common experiences; and with the deafened I feel sure that the suffering and the struggle are the same, no matter what the environment. One day a beautiful star appeared on my horizon. Faintly it gleamed at first, often obscured by the fleeting clouds of despair. That star was the birth of hope. Was it possible that my eyes could be trained to take the place of my ears? Was it possible that I could keep in touch with my family and friends in delightful intercourse? It was possible. I would try. All of this happened some years ago, but the star is still shining, leading me to new worlds to conquer and new lessons to learn. From the first I sought speech reading. Three things I count necessary to success along social lines: Courage, culture and a determined effort to master the art of speech reading. Limitations must be met and recognized, but I have yet to meet the situation where a courageous and frank attitude will not attract companionship. There is always a kind friend who will give a clue to the subject of conversation. Culture is an ornament that is especially becoming to those who have lost their hearing. A hobby is an excellent thing for the deafened. A determined effort to master speech reading is essential to all success. It is said that there are good speech readers and poor speech readers. I say we can all be good speech readers if we will persevere. And one of the most important things to do is to cultivate and develop the faculty of observation.

"The Deafened Woman in Professional Life"† was a paper read by Miss Julia E. Johnsen. She stated that the deafened woman in professional life assumes there is a handicap. This is only relatively true. It is true that we do not like, on account of our deafness, to approach people, to give them special

*Volta Review, November, 1922.

†Volta Review, November, 1922.

trouble, to place ourselves in a conspicuous or embarrassing position. Our sensitiveness or pride gets to the front. This is psychologic and not a handicap unless we make it so. We must not confuse any disadvantage with the fact of deafness, except as deafness contributes to the state of mind. What we need is clear thought and sensible conclusion that we will do whatever comes to us to do. Wrong mental attitudes are not held by us alone. Most obstacles can be overcome. We do not expect that deafened physicians, nurses, lawyers, and musicians, for example, can do exactly the same things as one who hears. Such training, or a strong attraction for special work, is too valuable not to be made use of. One might, for example, work with an assistant; or a physician might specialize in laboratory or X-ray work; a lawyer might turn from court to take up brief and record work, research, law librarianship, or even cataloging of legal literature. A musician might, like Beethoven, turn to composing, or to harmony or song writing; because music is not only a matter of physical sound, it is a matter of internal sound as well. I mention these things because one who has started out toward a professional career is apt to turn aside when deafness comes upon him, a fact which he may afterwards regret. In the case of a reporter, the ability to write well can be turned to authorship. We have a reporter who has become very successful in magazine writing. There are business standards to be met, rules, methods, resources or working facts to be learned. When these are learned the way is comparatively easy—that is to say, the work is related to one's capacity for the task in hand, and I think deafness is very little hindrance. This has been my own experience. My own work, however, is especially adapted, because it calls for very little communication. It is editorial work of a special kind and also reference library work. For the sake of the future it is better to expect too much of the deafened than to do the harm and lack the vision of not expecting enough.

A paper on "Compensations" was presented by Mrs. Robert C. Morris,* who said in part that he who has been afflicted with deafness only a short time is apt to be rebellious, and he

*Volta Review, December, 1922.

is skeptical that this universal law of compensation can possibly apply to his case. But he who has been deaf from infancy, through childhood and youth, and through middle age, knows that there is some soul of goodness in things evil. He knows that, as Emerson said, he can, like the wounded oyster, mend his shell with a pearl. When one of our five senses has become impaired the four others become strengthened beyond the normal. Intuition develops rapidly if we educate ourselves to know those flashes of knowledge which come to us intuitively, and the deafened may have through the life which must be lived in contact with other people, a more dependable guide than persons with normal hearing. The deaf person naturally becomes an accurate reader of human nature. They rarely make mistakes in judging men and women. He watches the lips of any new acquaintance, knowing that the eyes can be trained to deceive and other features may wear a mask, but the mouth is the only feature that cannot be made to camouflage the truth. Thus the deaf person selects his friends with infallible intuition. He sees in people important things that we people never see at all. His faculty for observation becomes the foundation for memory, reason, judgment and all kinds of culture. The deaf person not only has time to think, but he learns to concentrate. If he cannot carry on a conversation with John Jones or Wilbur Smith, it is just as well, perhaps, for the John Joneses and the Wilbur Smiths of the world seldom say anything that is worth listening to. If the sphere of the friendships of the deaf person is circumscribed, he may have quality if not quantity, and if he cannot become acquainted with a wide circle of friends, then he may turn his attention to Shakespeare and Emerson and Stevenson, friends who unite the whole world. If we cannot hear a lecture we may console ourselves with this thought: that the best of lectures always find their way into print, and 90 per cent of the lectures, so far as literary values are concerned, are not fit for print. Most of us can build a world of joy and accomplishments with such tools as we have. To be able to hear as the average person does is just to be an average person, but to be able to do something—to specialize—to do something better than the average person can do it, is to cause a thousand people who can hear to seek the path to our door. The

hard of hearing person is sympathetic, and the world needs that kind of understanding sympathy. Shakespeare said: "None can be called deformed but the unkind." We have come from many circles, we represent social and professional relationships of many kinds, and in this convention, perhaps for the very first time in our lives, we are among our very own people, for we are the people who understand each other so much better. Only you and I know the world in which we live, and I am proud to be numbered among your gallant company of the deafened. This very experience is one of the great compensations that deafness has brought to me.

These are but a few of the papers which have recently been written by highly intelligent people along the same lines, but time will only permit a few quotations from a remarkable article "On the Technic of Being Deaf," written by Earnest Elmo Calkins, a prominent figure in the literary and advertising world, and published in the January number of the *Atlantic Monthly*. It is unfortunate that we deafened can go so long without detection. We frequently choose foolishly, preferring to pass as slow, thick-headed, stupid persons rather than as the quick-witted deaf persons we really are. I have become a master of the art of being deaf. It is an acquired art. People are no more born with it than they are born bachelors. A bachelor is something more than a man who has failed of marrying; and the art of being deaf is something more than loss of hearing. When I asked Mr. Edison why he of all persons did not avail himself of one of the electric devices for making hearing less difficult, he said: "Too busy. A lot of time is wasted in listening. If I had one of those things my wife would want to talk to me all the time." Discussing a banquet at which it might be possible for me to be "seen," I said to a friend: "I'm not going to any more of those dinners. It's such a bore to sit there and not hear any of the speeches." "Not so much of a bore as if you heard them," was his feeling reply. After all, are not the deaf rather inclined to overrate the mere accomplishment of hearing? Audition is not without its drawbacks, and deafness without its compensations. For us, the noisy world is soft pedaled. My deafness beginning at fourteen years of age, my teacher, a woman of rare sympathy and helpfulness, would not permit me to fail and

insisted upon helping a discouraged boy to keep up with his classes. I still remember the enthusiasm with which she once said: "Earnest, I want you to succeed, not in spite of your deafness, but on account of it." There ought to be a book of short biographies of the famous deaf, telling how they met their problem. Such men as E. S. Martin, Thomas Edison, Sir Joshua Reynolds, Ludwig von Beethoven, the Earl of Chesterfield, Harriet Martineau, etc. We cannot draw any consolation from the belief that our condition is tragic. To begin with the first lesson and the hardest, it is imperative to admit that one is deaf—admit to oneself, and tell the world, and accept the penalties as well as the compensations. As some cornfed philosopher remarked, "Old maids is really the happiest—after they quits strugglin'." After all, the deaf are not deaf all the time; for they are not deaf when there is no occasion to hear. The deaf person should always prepare in advance for as many emergencies as possible. He should study the time table in advance, read the signs on the walls and in the train-shed. I have learned to set myself for awakening at any morning hour. Nothing can compare with a gainful occupation. He then mentions aids to hearing, lip reading, which is little short of marvelous, and then classifies the liabilities and assets in the way of recreation which a deafened individual may well commit to memory. On my index expurgatorious are:

Conversation in the best sense; the theater; lectures; public dinners and most private ones; music; social dancing; games like "What is my thought like?"; being read aloud to.

I have left: Books; pictures, moving and stationary; art, painting, sculpture, architecture and applied natural science; scenery; travel, on foot, train, boat, horse and motor; exhibition dancing, and all spectacles and pageants; games like golf and whist; nearly all hobbies.

Someone has said (boy, page Mr. Bartlett) that when God closes one window, He opens another a little wider. I have tried to help Him and swing my window altogether open. Among his recreations which he calls startling are painting, modeling, carving, fishing, digging in the earth, and play games. In consequence I have never been bored. I make models of ancient ships, which gives an excuse for hunting

old books and prints to learn how they looked and were rigged. I have a colony of bees, which are sufficiently amiable to permit me to take out the brood combs and find the queen, when there is someone to see me show off and exclaim: "Don't they ever sting you?" I also battle with beetles and worms, for the satisfaction of raising a few of the varieties of roses. If I play a rotten game of golf, it is not because I am deaf. There is no reason why a deaf man should not be a very good golfer. With me it is, perhaps, because I enjoy a walk as much with a blackthorn stick in my hand as with a mashie. Do not get the idea that all this means dispensing with friends. Friendship is not conversation. The things the deaf can do to reclaim the waste places of life, and find happiness in doing, have another rare quality. They are a substitute for conversation in a quite different way. They enable him to account for himself to others, to acquire a new interest in the eyes of his friends, to win a consideration that his amateur performance as a listener will not give. Thus I find myself at fifty-four busy and happy, with a very satisfactory "expectancy" allowed by my insurance company, with a life packed full of the most exciting and enthralling things to do, and wondering whether I am going to have time—even if I realize that expectancy—to do them all.

ORGANIZED SOCIAL SERVICE FOR THE DEAFENED.

It is obvious that any sustained effort to benefit deafened people along social service lines must be through proper organization. Formerly no institution work was attempted for the social and economic betterment of the deafened, except through institutions maintained for deafmutism. It so happens that the psychology of the deafened person with speech differs so widely from mutism, or deafness without speech, that it becomes well nigh impossible to carry on any social service or educational work which contemplates a combination of the two conditions. This fact has been fully proven and needs no further discussion. The balance of this paper will have to do chiefly with social service work for deafened people or those with speech.

During the last ten years organizations have come into existence with the purpose of solving these problems. These

organizations have assumed various names, like leagues for the hard of hearing, speech reading clubs, etc. Community work for the deafened would seem to be a suitable general term under which the various subdivisions of social service work may be outlined. The ideal organization, in my judgment, is one that would be based largely upon the club idea. First, a permanent home, suitable for carrying out the necessary activities. Second, with trained social workers in charge. Third, independence and self-maintenance on the part of the members, so far as the actual club expenses are concerned. Outside help should be secured covering the expenses of education, vocational training, employment, recreation, charities, research and preventive treatment. Unfortunately, during the earlier stages nearly all organizations have been obliged to receive donations in order to successfully become established. Let us briefly define an ideal community center for the deaf. Ideally, such a center should undertake to do for deafened people service similar to that which is so successfully done for the blind. In New York City the community center for the blind centers around an institution known as "The Lighthouse for the Blind," which is a large home or clubhouse, which contains rooms for the various departmental activities. This would be an ideal arrangement for a community center for the deafened, which should primarily be a clubhouse where deafened people could carry on the various activities. Such a building might include rental quarters for private teaching of lip reading. In some cities in America the Municipal Community Chests have provided the headquarters and equipment. In the formation of a local unit three types of individuals should, if possible, unite—the otologist, the deafened educator and the deafened social workers, in a combined effort to rouse the deafened man and woman out of despair and make them over again into alert, happy, hopeful and self-supporting citizens. Just get a group of the deafened together and organize. The American Federation of Organizations for the Hard of Hearing is prepared to furnish the necessary organization literature.

EDUCATIONAL (LIP READING TRAINING).

Inasmuch as Mrs. Nitchie, in her paper, will cover the entire field of lip reading education, we will be content in merely

urging this procedure and suggesting that otologists make it a routine matter of advice and counsel to their patients. Even though hearing devices are employed, lip reading should be developed in every case, so far as the individual is able to avail himself of this valuable art. It is particularly urged that in the early stages of deafness patients commence the study of lip reading in order to become proficient before the time has arrived when it becomes actually necessary.

EMPLOYMENT BUREAU.

One large department of such a service should be the employment bureau, which should be in charge of trained vocational counselors and placement workers, capable of advising applicants for positions in accordance with the type, degree and progressiveness of his deafness, his physical condition, his education and his temperament. The employment workers in the New York League for the Hard of Hearing make a careful study of each individual case, making use of a card index system which becomes permanently available, and which covers every phase of the applicant's condition. The following are among the questions asked:

1. The age of the individual at the time he is forced to consider rehabilitation.
2. The degree and the type of deafness at the time he recognizes the need for rehabilitation.
3. The finances of the individual and his dependency.
4. The willingness of the individual to accept the aids, such as lip reading, hearing devices, etc., that are available.

The placement workers are obliged to overcome, first, the inadequate facilities for training in a wide range of occupations; and second, the unapproachability of employers. The rehabilitation of the deafened junior is a comparatively simple problem. He is trained in lip reading, equipped with a hearing device, if necessary, and then trained for a vocation where any increase in his deafness will not be a handicap. These occupations include most forms of machine operating, all branches of the sewing trade, all of the agricultural pursuits, some of the professions, and many kinds of business. The exceptions include telephone, teaching (except lip reading), telegraphy, stenographic work, salesmanship and cashiering.

The league has advised training to some of its younger applicants in printing, show card writing, embroidery machine operating, designing and office machine operating, such as the billing machine and multigraph.

The rehabilitation of the person who has engaged in some definite occupation for many years is a more serious problem. Thirty-seven per cent of all the persons who apply to the bureau are forced to change their vocation. For them, advice regarding the utilization of part of their former training and experience must be given, suggestions for proper training and the place to take it, and information regarding those occupations which might be harmful. To those with subnormal hearing any remnant of hearing is most precious; hence, occupations surrounded by unhealthy conditions, such as are found in refrigerating plants, subways and tunnel construction, must be avoided. Positions requiring standing must be avoided by deafened persons who are subject to vertigo. Let us cite the histories of a few cases that have been handled by the bureau during the past year:

1. A nurse with slight deafness. Sought advice from nurses' agencies, doctors and friends. Sought advice at the League. Trained in lip reading, told to give up nursing, given a job where she would not be too fatigued to study lip reading at night; arrangements made for training through the Bureau of Rehabilitation of the State Department of Education, in X-ray operating. Secured a position in a hospital. She wrote: "I was prepared to have a hard time of it, but now scarcely feel that my job is work. I have found it good to combine X-ray with laboratory work and have taken a course in bacteriology. It is not only working, but also the planning to do more that makes me feel a bit more useful again. I am glad to tell you that I can do the work and can also enjoy it. Please accept \$5.00 for the League from my first salary.—N. G."

2. Young man, 22. Total deafness after mastoid operation. Clerk in insurance firm before. Advised him to learn all about office machinery. Finally took advice and secured employment in three weeks' time.

3. Man, 50. Very hard of hearing. Uses a hearing device. Yacht captain for seventeen years with the same employer. Lost job not because of deafness, but because man had given

up yacht. We have advised this man, who has some savings, and a wife with perfect hearing, to open up a small shop in a suburb, such as a hardware store, where the counter work could be done by his wife and the handyman work and mechanic work be done by him. In order to convince him that people of normal hearing would accept him on the strength of his ability in spite of his deafness, he was placed in a job as a mechanic for a time.

In many instances a new occupation has to be learned, and the applicant must be given a course in vocational training. In such cases the placement workers, having made a complete study of the individual, are able to advise the kind of occupation for which he is fitted. He is then given such schooling as may be necessary to prepare him for his new job.

An important element in the employment for the deafened is the ability of the placement workers to convince employers of the availability of this type of employee. In the New York League this has been so successfully done that various employers have shown their appreciation by calling for recruits from deafened sources. He is told that the deafened man concentrates better than the hearing; that he is more efficient at general routine work; that he accomplishes more work in less time, for the reason that he does not engage in conversation going on about him. Furthermore, he is more appreciative of his job because he recognizes his limitations and does not seek constant change. In short, his redeeming features, to be mentioned to his employer, are as follows:

1. Highly developed power of concentration.
2. Keen observer.
3. Quick mental movement.
4. Does not waste time in chatter.
5. Remains in position longer than hearing individual.
6. Master of detail.

Of late the demand of employers has been so great that our placement officers are carrying a newspaper advertisement urging deafened people to apply for positions.

VOCATIONAL TRAINING.

Vocational training whereby deafened people may be taught new occupations should be urged in all cases where the victim can no longer follow his usual vocation. In the larger cities

there are special organizations for vocational training along all lines of endeavor. In New York City the Bureau of Rehabilitation of the State Department of Education pays for the vocational training of our deafened individuals in the existing schools of the city. Thirty-four States in the Union offer rehabilitation facilities to the physically handicapped. It is up to the organizations for the deafened and the otologists to make use of them for their cases. In smaller cities the organizations should undertake to find training opportunities for their people. If possible they should provide scholarships in trade schools that would cover traveling expenses and board if necessary. Above all, avail themselves of the city, State and Federal facilities offered and demand them where there are none. Whatever else he may do in preparing for a new job, he should certainly persist in his lip reading studies.

HEARING DEVICES.

Dr. Walker is to discuss the question of mechanical aids to hearing; hence I shall confine my remarks upon his subject to a few brief comments regarding certain important information recently gained. In general, it may be stated that great progress has been made during recent years in the development of hearing devices, particularly in their special adaptation to varying and different uses. For instance, until recently, the executive secretary of the New York League for the Hard of Hearing, Miss Annetta W. Peck, has been entirely unable to make use of telephone conversation. About one year ago she found that by using the phonophor applied to the telephone receiver she was able to hear telephone conversation perfectly. The writer converses with her frequently and has been amazed at the power of the little instrument. The Phonophor Company produces four or five varieties of phonophor, but the one most effective for telephone service happens to be the cheapest one of the lot. Stethoscopes with large mouthpieces (accumulators) can be purchased at Knauth Brothers, New York City. These might be of service to hard of hearing doctors in examinations of the heart and lungs.

Mr. Storey's comment, previously referred to, calling attention to the fact that hearing people frequently approach the deafened when they see them using the hearing device, is worth repetition at this time. The various manufacturers of

hearing appliances are more than willing to undertake any problem which tends to improve their quality and effectiveness. A recent experience has brought to light a new phase in the hearing device problem which promises to bring untold happiness to many deafened individuals. This knowledge may be in the possession of many in this audience, but the writer's knowledge is of very recent origin. Almost by accident some deafened people have been induced to test the radio with wonderful results. In one instance a deafened man was urged to try it, but for days and weeks refused to even attempt to make use of the radio, only to be enraptured by his first day's experience with this marvelous invention. His letter written at the request of the writer will best tell the tale.

"Dear Dr. Phillips—Knowing of your great interest in the cause of humanity, and especially in those who are hard of hearing, I desire to tell you of my wonderful results from the use of the radio.

"I have been deaf for 45 years—have been deprived from intercourse with my fellow men—deprived from the pleasure of attending lectures, concerts, church. Now, however, that, thank God, is ended, and also ended for all those who are similarly afflicted.

"My outfit is not a very wonderful one—made by the Radio Instrument Company of Washington, D. C., and makes use of six vacuum tubes, yet by 'plugging' in the first stage of amplification I hear perfectly.

"I had had my radio but a few days when on Easter Sunday I listened in to the service conducted at the West End Presbyterian Church, heard distinctly, plainly, clearly, the opening organ voluntary, solo and quartet singing, reading of the lessons, prayers, a wonderful sermon, installation of new members and the benediction, and please believe, it was, to me, a God sent benediction. I say it manfully and without shame or fear of ridicule, the tears of joy streamed down my face. Last Sunday I heard the entire service at St. Thomas' Church—in the afternoon the services conducted by the Bedford (Brooklyn) branch of the Y. M. C. A., by the Rev. Dr. Cadman, who stated there were nearly 1,000 men in attendance, and when this congregation sang that glorious, inspiring hymn 'Onward, Christian Soldier,' the music being played by the

Glory Trumpeters, you can imagine the effect it all had on a poor soul who has spent forty odd years in the 'silent land.' In the evening I listened to a concert by the Capital Theater Orchestra, and the ending of a perfect day was a fine organ concert. I want to add that one number of the theater concert was the 'Melodie' in F, by Rubinstein, played as a 'cello solo. I've heard, off and on, parts of this composition all my life, but never before the entire piece, until it all came to me over the wire, the deep bass notes of the instrument sounding like a beautiful pipe organ.

"I have, so to speak, by means of the radio, found a new heaven and a new earth and am just beginning to lead a normal life. God grant me many years of life in order that I may enjoy, to the utmost, my new found joy and happiness.

"If all this wonderful salvation has come to me it can also come to others in affliction, and I shall appreciate it highly if you will tell me how best to go about spreading the gospel of Saint Radio, as I cannot keep it to myself, but want to shout it all from the housetops for the enlightenment of the world.

"Please feel at liberty to use this epistle in any way desired, so long as it will do some slight good for those who may be afflicted and whose souls are crying for help. Your friend,

"WARREN POND."

Mr. Warren Pond is at the head of the well known firm, William A. Pond & Company, music publishers, of New York, and is evidently a case of otosclerosis.

Since receiving this letter several people with varying types of deafness have been requested to make tests of the radio in an attempt to determine just what types of deafness may be reasonably expected to make use of the radio. The following letters relate the experience of other people:

"Dear Dr. Phillips—You asked me to give you some account of my experience in listening to Mr. Pond's radio. I have tried it twice, once on April 24, and again April 29, when my father went, on Mrs. Pond's invitation. His experience is also interesting; so you have mine, otosclerosis of 38 years' standing, and my father, C. C. O. M. of 35 years' standing. He is quite hard of hearing now, while I have practically no hearing at all, unless hitched to the most powerful devices made.

It is impossible in my own experience to separate the emotional and imaginative from the scientific; but I think you are as much interested in the one as in the other. When, about ten years ago, I dismissed my last pupils in piano and harmony, I slammed the door on my old life and plunged into earning my own living in an entirely new field. So completely have I forgotten that I was ever a musician, and identified myself with social work for the deafened, that I have wondered whether there really was sufficient magic in things heard to justify people in spending hours and hours of precious time sitting around a concert room or an opera house. You can judge therefore of the emotional effect of hearing over the radio the tremendous and thrilling voice of my own beloved piano, not painfully incomplete, but full, rich and satisfying as in the early days of my deafness before I ever dreamed that music would have to be left behind and its glories translated into some other form of human activity and service.

"The repertoire of the piano does not change much, and from old experience I could hear all variations of touch and even the errors of the young player's performance. I heard all announcements surprisingly well, getting names better than titles, and later, in the baseball reports, the names of clubs better than those of individual players.

"On the second occasion I heard, besides announcements and short speeches, songs by sopranos, a tenor and a baritone, orchestra and organ. The latter was particularly successful.

"On both occasions I found my head noises slightly increased—a sort of escaping steam sound—and that my head ached slightly for a time, but my speaking voice sounded clearer to me and my hearing definitely better. Of course, hearing in my case does not mean the naked ear, but hearing familiar voices at their customary pitch and volume with the same hearing device as usual. After two days this continues.

"My father, on the other hand, heard almost too much, just as Miss Samuelson did, and as he does not like the electric hearing devices much at the best of times, he was making all sorts of little experiments in bone conduction, which is more comfortable to his thinking. But the day following he was much deafer, so that we had to raise our voices very much more than usual—in fact, it was all we could do to make him

hear. This was better the next day. However, he is going to be a fan, I can see. Hoping that this will be of some use to you, I am, sincerely yours,

"ANNETTA W. PECK."

"My dear Dr. Phillips—Here's a statement of my experience with Mr. Pond's radio. We listened in on piano selections, several soprano solos and a male voice announcing commission market quotations and baseball scores. I found the speaking voice by far more agreeable. When the radio was tuned at a pitch comfortable for Miss Peck—that is, so that she could hear the piano as an actual piano—it was a most violent noise and tin canny. The vocal selections were ever so much better. During one selection there was a crossed wire, and I could clearly hear the vocal selection with a jazz band and coon shouter. It seemed as if I was hearing one in one ear and the other in the other. When the radio was tuned so that it was comfortable for Mr. Pond it was agreeable to my ears. (I guess that you will agree that Mr. Pond is a bit more deafened than I.) The voices were all clear and distinct.

"The day following I was walking through the tunnel outside of Gimbel's. I heard a phonograph just as clearly as if I stood alongside of it. I investigated. In the store near one of the entrances I found a radio with the horn attachment. I should say that I heard the music (as much, which is rarely true of a naked phonograph) at a distance of at least 150 feet through walls and windows. I don't hear our own victrola at home unless I am in the room in which it is playing.

"The possibilities of radio as a reeducator of hearing seems great to me, but raises one great question in my mind. I really am a fan and wouldn't want to take the joy out of the game for the world. Very sincerely yours,

"ESTELLE E. SAMUELSON."

Miss Annetta W. Peck I know to be a case of otosclerosis and her father to be one of chronic catarrhal deafness. These experiences certainly serve to determine the fact that the radio has brought marvelous possibilities of experience for our deafened friends. It promises to become a source of educational value, even in the schools for deafmutes. So enthusiastic have a few members of the New York League become that the whole place is being installed with radio equipment for the

benefit of its members. Several have already remarked that they propose to attend church every Sunday morning and evening at the League headquarters and to listen to lectures and concerts at other times.

Some of our workers have found much benefit from an appliance called the Auricles, manufactured by the William V. Willis Company of Philadelphia. This instrument is quite similar to the English made instrument known as the Aurolase Phone, but possesses the added advantage that it can be purchased for about \$10.

RECREATION AND AMUSEMENTS.

It is surprising how much real pleasure may come from organized efforts to promote certain types of amusement and appropriate recreation measures for deafened people, particularly in groups. Ordinarily, the deafened person gets his chief amusement from books, moving pictures or from some fad. It is possible, however, under trained leadership to develop a variety of amusements for men and women of mature years, such as card parties at headquarters, for which clubhouses should provide opportunities. Younger people become very much interested in theatricals of the pantomime type, and when possible they are exceedingly fond of dancing. Recreation in the form of club hikes, with lunches, botanical, nature and other available studies.

PREVENTIVE DEAFNESS.

Within the domain of social work, under otologic supervision, can well be placed opportunities for studies along the line of preventive deafness. Naturally these studies will have to do largely with child life. Dispensary and hospital treatment for the deafened has up to date failed to conduct specific investigations along the lines of preventive deafness in children. It is true that certain well known causes are carefully attended to in the large clinics. By this I refer to the removal of tonsils and adenoids and similar measures, but to give a specific study to children who already show signs of deafness and to treat these children with the sole purpose to prevent further inroads upon the hearing function has never been generally adopted. Dr. James Kerr Love of Glasgow*

*Volta Review, November, 1921.

has done some pioneer work along this line, and Dr. Franklin W. Bock has attempted the same measures in the Rochester public schools.

Since October, 1922, it has been attempted under the auspices of the New York League for the Hard of Hearing. The plan has been about as follows: No children have been subjected to examination and treatment except those who have been secured under League auspices and by our professional workers. In every instance full records are obtained at the League covering the name, age, home conditions and all other possible information is obtained and recorded. This information is not only recorded but filed and a duplicate record card is sent to the doctor. The following is a sample of the card:

CLINIC FOR THE PREVENTION OF DEAFNESS IN CHILDREN
NEW YORK CITY

Date.....

Name

Address

Age

Degree of Deafness

Cause of Deafness.....

Speech

Illnesses

Deafness, first notice.....

Head Noises

Earaches

Tonsils and Adenoids.....

Where treated

Deafness in family.....

Parents' name.....

School

Grade

Teacher

Remarks

Registered by:

The New York League for the Hard of Hearing, Inc.
126 East 59th Street

The patient is then given a complete otologic examination by competent otologists. This examination is supplemented by such other forms of examination as the symptoms may suggest. In some instances even a Wassermann test has been made. The child is then placed under treatment—nasal and pharyngeal abnormalities and diseases eradicated, advice and counsel is given as to diet and home life. The prevention of colds and proper aural guidance during and after colds. So far no efforts have been made to enter the field of pathology. Furthermore, the number examined up to date is too small to enable us to form definite and permanent conclusions. In all, about 60 children have been under observation and have been given a total of about 500 examinations and treatments. A preliminary report will probably be published in the near future. Suffice it to say the results of these examinations and treatments have more than warranted the efforts which have been put forth, and it would seem that the time has come for the opening up of departments for research in the prevention of deafness in our ear hospitals and clinics. Furthermore, it should become one of the leading features of social service work in the field of otology. Particularly, in our very large cities for the treatment of ear diseases, there should be a distinctive department devoted entirely to the subject of preventive deafness. I venture to report a recent case coming under my observation which serves clearly to show the need of a more careful study of hearing conditions in children, with a view to the prevention of deafness, and second, to the proper treatment of children showing symptoms of defective hearing. A girl ten years of age showed symptoms of gradual loss of hearing for about two years. The mother had taken her to one or two general practitioners, who failed to discover any cause for the affection. In school she had commenced to fall back in her studies, and the teacher had finally advised the mother to place the child in the public school for the deaf. It is evident that no competent otologist connected with the school system had ever examined this child. It was found that her hearing was so defective that it would be impossible for her to continue her studies by regular school methods. Upon examining the canals they were both found to be tightly packed with cerumen, and after its removal she heard as well

as ever. This case may be a rare exception, but it serves to emphasize the laxness in our present methods of examining school children and emphasizes the fact that there is a wide field for investigation along the lines of prevention in the domain of aural diseases.

This I have briefly outlined various phases of social service work which may well be attempted by individual organizations. After two or three years of rather intensive study of the problem as undertaken by the few organizations in the larger cities, I became convinced that this type of enterprise should be encouraged by the formation of a national organization, for the following reasons: First, to provide a central body which would have the control and management of an annual convention in the interests of local organizations. Second, for interchange of views and for development of methods, and to encourage the formation of local organizations throughout the country. This organization is now on a well founded basis, under the title of "The American Federation of Organizations for the Hard of Hearing." The fourth annual convention was held in the city of Chicago, June 18, 19 and 20. The third annual convention was held in Toledo in June, 1922, and over 250 delegates registered from various parts of the Union, all the way from Boston to San Francisco. At the present time fifteen constituent bodies are in affiliation with the national organization, and many more are making ready to join or are in the process of formation. There are two foreign local associations—one in Glasgow, Scotland, and one in the city of Prague. The *Volta Review* has become the official organ of the Federation, which has its headquarters in the *Volta Review* building in Washington, D. C.

In conclusion, I would say that social service work for the deafened offers a wide field of actual service to humanity on the part of the otologist. Every large town or city should have an organization to carry on this work. The otologist, recognizing the timidity of the deafened in public matters, should lead in the formation of these local units. Many of the local organizations throughout the country have come about in just this manner. Upon entering this field the otologist opens the door of his life to the very highest realm of actual service to humanity. It calls for unselfish expenditure of time

with little to be gained along the line of financial emoluments. For in the main he must give of his time freely. It is a disinterested service and brings little fame. It is the fulfillment of the provisions of the Hippocratic oath in its very highest sense.

NOTE.—Comments herein made from papers of Mr. Starling W. Childs, Mrs. George L. McAlpin, Mrs. Julia E. Johnsen, Mrs. Robert C. Morris, Mr. John de Raismes Storey and Mr. Earnest Elmo Calkins are practically in the exact language of the individual authors.

AIDS TO HEARING.

By GORDON BERRY, M. D.,

WORCESTER, MASS.

On the 4th of August, 1922, thirteen million telephones were silenced while the nation mourned one of her greatest. Poor and potentate the world around sent messages of sympathy and condolence. As the sun was setting on Beinn Bhreagh, the highest spot in Eastern Nova Scotia, there in the solid rock the body was laid to rest while the stillness was broken by the singing of Stevenson's "Requiem":

Under the wide and starry sky,
Dig the grave and let me lie;
Glad did I live and gladly die,
And I laid me down with a will.

Five months have passed, and on the 3rd of this month (January, 1923) his widow, alike the cause and inspiration of his wondrous accomplishments, went to join him. Increasingly as the years swing around will man realize his debt to Alexander Graham Bell and Mabel Hubbard Bell. To some who are intimate with his manifold activities it seems that not in his remarkable geographic research, not in his exhaustive statistics on heredity, not in his pioneer work with aeroplanes, not even as the inventor of the telephone, but in his constructive championing of the deaf, lies his first claim to the gratitude of posterity. The proximity of their deaths, and their intimate association with all progressive effort for the deaf make it fitting that we refer to these two and their early struggles. We can thus render homage, and at the same time through their lives review the history of work for the deaf during the past fifty years.

Dr. Bell's grandfather gained fame in Scotland as a teacher of elocution and speech training. His father, Mellville Bell, invented the famous "universal alphabet" now used in teaching phonetics. It was Dr. Bell's boyhood enthusiasm which cre-

ated a rubber skull that could say "mama" and that so trained a pet dog that when Bell's fingers pressed successively on different parts of its larynx, the dog could be heard to say "How are you, grandma." When (at the age of twenty-four) Bell first came to Boston in 1871, Miss Sarah Fuller was at the head of the Horace Mann School. She early asked him to teach her workers. Then he spent three months at the Clarke School for the Deaf and seven weeks at the Hartford School. He rapidly became an authority in speech training. To his Boston school came young Mabel Hubbard, whom he later married.

A few years previously (in 1864), at four and a half years of age, this little girl had become totally deaf from scarlet fever. Her father learned that the Germans were successfully teaching their deaf by the oral method, but that in this country only the sign language was used. Mabel seemed destined to lose her speech as well. Here in Rhode Island Governor Henry Lippitt had a deaf daughter, Jennie, whom the mother had been teaching by the oral method for six or seven years. With Mrs. Lippitt's encouragement and advice, Mrs. Hubbard now began to teach Mabel, and so successfully that at six years of age she and her friend Jennie Lippitt were brought before the legislators on Beacon Hill, where the question of the founding of an oral school for the deaf in Massachusetts was hanging in the balance. The Solons could not but yield to the convincing proof these two little girls gave, and in 1867 a charter was granted the Clarke School for the Deaf at Northampton, the pioneer of the oral schools in this country; for the Hartford School, founded in 1817, and other more recent institutions for the deaf were all using the sign language method which Gallaudet had brought over from the Abbe Sicard's school in Paris. In the meantime Isaac Rosenfeld had been seeking aid for his deaf child. The final result of his efforts was the opening of the "New York Institution for the Improved Instruction of Deaf Mutes" in 1867, and nine years later the "Rhode Island Institute for the Deaf" was founded here in Providence. To Gardner Green Hubbard, Mrs. Mary A. Lippitt and Isaac Rosenfeld belong the credit for sponsoring these splendid institutions, but not more than to Harriet B. Rogers in Northampton, Sarah Fuller and Mary H. True

in Boston, J. W. Homer in Providence and Bernard Engleman in New York, the pioneer teachers who built the foundations of our structure.

The second chapter comes with Dr. Bell's efforts to help these deaf children. He had found by tests that many had some hearing. By utilizing this little, he was much helped in getting them to place their sounds correctly. He used speaking tubes with profit and was anxious to discover some electrical means of magnifying sound that he might still better reach them. With Dr. Clarence J. Blake's help he fastened a stylet to the ossicles on a model of a human ear and was able to obtain a record on a rotating drum of the membranous oscillations made by the spoken voice. He called this the phonautograph, and had the deaf children use it. He found that a small resonator box containing tense violin strings when worn next to the child's chest would give tactile warning when a bell or horn of an approaching vehicle on the road spelled danger. Then he became convinced that some resonator might be so adjusted as to cause an undulating current in an electric wire, and that the reversing of the process would pick it up in the receiver end as duplicated sound waves. This was his initial conception of the telephone. At about this time (1874) Bell was working as a side issue with his "harmonic telegraph," in an attempt to send six or eight Morse messages on a single wire at the same time without interference. The mechanical work was being done by Thomas A. Watson. With characteristic perseverance, Bell worked through that winter and the following spring. He was using springs of different tension, something like tuning forks, and trying to have the electrical wire transmit a selective impulse as sound to similar springs in an adjoining room. Watson describes that momentous afternoon of June 2, 1875. "We were hard at work at the same old job. One of the transmitter springs I was attending to stopped vibrating and I plucked it to start it again. It didn't start and I kept plucking it, when suddenly I heard a shout from Bell in the next room, and then out he came with a rush, demanding 'What did you do then? Don't change anything. Let me see!' The make-and-break points of the spring had become welded together, creating just the necessary electric circuit for transmitting sound." Fortunately

it was the genius Bell who was in the other room and who heard the faint ticking on his end of the wire. There was born his telephone. A similar good fortune in 1900 destined that Lee DeForest should be working near a Welsbach burner when testing electrolytic detectors for wireless signaling. He noticed a brightening and dimming of the gas light as the spark transmitter was operated. From this came the audion amplifier, which permits telephonic conversation across the continent, enables a vessel to come into New York Harbor in a fog, and promises to play an important part in electric hearing devices of the future.

It was not many days before Watson's fevered activity under Bell's direction was able to make the crude transmitter through which the first telephone message was delivered—"Mr. Watson, please come here. I want you." Then came endless experiment and adjustment. Finally they were ready to make an outside test. Permission had been gained from the Walworth Manufacturing Company to connect up with their private telegraph wire two miles long, running from Boston to Cambridge, and on October 9, 1876, Watson first heard Bell's historic call "ahoy, ahoy," over the long distance telephone. Perfecting the telephone consumed so much of his time that Dr. Bell had to give up other activities for the time being. But his interest in the cause of the deaf was just as active, as witness his letter to his mother in this same year (1875) when convinced of the success of his invention: "Now we shall have money enough to teach speech to little deaf children." And it was Mr. Hubbard and Mr. Thomas Sanders, fathers of two of his deaf pupils, who furnished the financial backing to start the telephone. Thus inseparably is the telephone connected with deafness. And later this same telephone was to be the basis for the electric hearing devices which we are now using.

With the establishing of the Bell Telephone Company and the clearing up of legal difficulties following infringements of his patents, Dr. Bell was free to enter such fields of endeavor as his desire directed. In 1878 he moved to Washington. Here he was always ready with voice and pen to enter the lists vigorously in behalf of any enterprise that tended to

ameliorate the isolation of the deaf or better their training. Here he conducted his extensive researches in hereditary deafness. Of peculiar interest was his study of the colony at Gay Head on Martha's Vineyard. The excessive deafness among the inhabitants there, together with accurate historical records of their consanguineous marriages, led to his important conclusions against the marriage of the congenitally deaf. He called together the first convention of oral teachers for the deaf, which met in Worcester, Mass., in January, 1874. This group kept itself intact under his leadership and was stimulated by his summer gatherings at Lake George until it won recognition at the twelfth convention of the American Instructors for the Deaf (sign language group) and was incorporated in 1890 as "The American Association to Promote the Teaching of Speech to the Deaf." Dr. Bell was its first president and endowed it with a personal gift of \$25,000. When the French Government awarded him the Volta Prize of \$10,000, because of his discovery of the telephone, he founded the Volta Bureau and gave it this money for the equipment of the Volta Laboratory. Here were invented the graphophone, wax disc records and an improved phonograph. This prize gift and his subsequent donations made a total of about a quarter of a million dollars. In 1909, at the founder's suggestion, the Volta Bureau with its endowment was presented to the "American Association to Promote Speech to the Deaf." Today this Bureau publishes the *Volta Review*, has a library which is the most extensive repository of literature concerning phonation and the deaf in existence, serves as a rallying point for new enterprises for the deaf and as a patron of all who will conduct research in their behalf—in a word, is the central agency for the deaf in this country.

With this cursory review of the early work for the deaf, let us turn to the present day aids to hearing that Dr. Bell's efforts and those of others have contributed. The subject naturally divides itself into a consideration, first, of the mechanical devices to aid hearing that are on the market; second, lip reading as a very important help; third, of the cooperative work now being done in this country for the deaf.

There are many mechanical contrivances purporting to make the deaf hear. They all have something in their favor.

First, there are the various forms to be placed in the auditory canal to fill in a perforated drum membrane. Considerable skill is attained in applying the piece of rubber or membrane. By actual test the hearing may be better after its insertion. Such appliances are to be condemned because they are not intelligently used, and because the unsuspecting victim almost invariably injures the canal or aggravates or creates an active suppurative process. The second type evidences itself in some form of modification of the hand cupped around the ear to facilitate hearing. The Globe Phone Company advertises nine forms in its circular, all of which are on the basis of a horn or tube which will catch a larger volume of sound than the unaided ear and will convey this directly through a confined air channel to the external auditory meatus. This type has the advantage of transmitting sound, exactly as it is received without accentuating certain types of sound and lessening others, and is found particularly serviceable in the aged deaf. In this group may be included quite a different mechanism, which relies on bone conduction for hearing. The best is a hard rubber fan held against the teeth. Sound impressions are caught on it and carried through the teeth to the skull and so to the internal ear. The third type is being steadily perfected and, by reason of its finer control and better adaptation, is gaining supremacy. I refer to electric hearing devices.

Priority in manufacture of the first ear phone appears to be in doubt. I am informed that C. W. Harper first brought out his Oriphone in Boston in 1902, and that Miller Reese Hutchinson of Mobile, Ala., first produced the Akouphone in the same year, changing the name later to Acousticon. The Globe people were also pioneers. These instruments have been made in many sizes. The smallest of which the writer has heard had a receiver which could be inserted almost out of sight in the ear canal, a battery one inch long and three-quarters of an inch wide, and a transmitter the size of ten cent piece which could be worn as a lapel button. Such small sizes are impracticable. An enumeration of the different standard ear phones on the market in this country will give a concrete idea of the growth of this industry. They are the Acousticon, the Gem, the Globe, the Harper, the Magniphone, the Mears, the Midget Phonophor, the Opera Type Miss Lucy M. Bruhn

brought last fall from Germany, the Portophone, the Potter, and the Williams Articulator.

The mechanism of the telephone and of these instruments depends upon what might be called a loose connection which has been gained by the insertion of many carbon granules or balls into a continuous electric circuit. These are enclosed in a chamber or in many little cups, and the movement of the diaphragm against which they rest makes a larger or smaller number of electric contacts among these balls. This varying resistance gives an electric fluctuation: Bell's "undulating" current. Occasionally a carbon ball gets caught, making a permanent contact, with a consequent hissing sound. This and other imperfections manufacturers are trying to correct. The battery for furnishing the electric current has been improved and simplified; unnecessary parts have been eliminated; the electric cord has been perfected. One worker (Charles W. Harper) spent two years in determining the best material for the diaphragm. The war has contributed its quota. Earl C. Hanson, by experimenting with the audion amplifier in his wireless work, was able to help men at listening posts hear the enemy's plans, use a tree as a wireless receiver, enable a flier to get messages from the ground. His deaf mother had made him anxious to make some contrivance to help her, as those already existing did not seem to serve. With the cooperation of the Western Electric Company, which controls the audion patent, he made the Vactuphone for the Globe Company and thus introduced a new principle into the science of ear phone manufacture.

An ear phone transmitter lends itself to considerable adjustment. Two factors are present. The writer is told that the size and texture of the diaphragm determine the amplitude of its wave length, while the stiffness of the diaphragm and the controls at circumference and center determine the amount of its in and out swing. The first factor regulates the pitch which is transmitted through the cord to the receiver and the ear; the second determines the intensity of that sound. As we know experimentally that certain patients, though deaf, can hear far more clearly at certain pitches than at others; and on the other hand that where there is a partial nerve deafness superimposed a greater intensity both tires the ear and

drowns out the sound; it is clear that these two factors of distinctness or pitch and volume or intensity must be intelligently controlled. Does not this suggest where the otologist may contribute by determining the degree and type of deafness? In the research laboratory of the Western Electric Company one can so carefully test an ear with their graded sound producing appliances (such as the audiometer) as to chart the acuity of hearing for differing pitch and volume.

How much gain can a deaf patient expect from a properly adjusted ear phone? A conservative estimate would triple the range of hearing in the average catarrhal case. This is a real gain. Manufacturers and deaf alike say that one must train himself to use the instrument. Certain types of noises are much magnified, others are scarcely audible. The user learns how to adjust the rheostat, dimming the loud noises and increasing the soft. Adventitious noises which the instrument accentuates he learns how to pay no attention to. In nerve deafness these adventitious noises trouble more, and here the ear phone offers no aid but jangles and tires the ear. Where there is a catarrhal as well as a nerve deafness, an instrument which accentuates detail without undue volume will help the catarrhal phase and not tire the nerve phase. It is claimed that these instruments improve hearing. There is logic in this contention. We know that disuse of a nerve causes its atrophy.

What is our custom when we are persuaded that we cannot further help a deaf patient? Do we ask him to see whether lip reading will help and bid him go to the store and secure an ear phone for one week's trial? Can we not be more accurate and definite in our recommendations? One manufacturer suggests the possibility of grading the instruments into standardized groups. He would make some device whereby the otologist can test the patient in order to discover which ear phone will fit the given case. We could then prescribe an instrument as the oculist prescribes glasses. The ear phone is still in its infancy. When it reaches full stature it is conceivable that such an Elysium awaits the hard of hearing patient.

We have mentioned how lip reading for children received its first impetus in America. Schools for deaf children sprang up in every state, and a tremendous amount of good is being done by them. It is not generally realized that from an institution

like the Clarke School for the Deaf an average of at least one goes out each year to enter high school on equal terms with the hearing children, and that one-half of these go on through college. The handicap is present, but with the use of other faculties than the one impaired sense of hearing, graduation with honors is attained, and a successful life in business or some other pursuit is the result. It is of the adult deaf that the writer would speak. This is a different study. Education has been acquired, the power of speech has been fully developed, contact with the world and one's fellow men has been intimately established. That contact must be preserved when deafness has come. Is the ear phone the only means?

Chance actively identified the writer with the Army Lip Reading School for the returned war deafened soldiers during the most of its fifteen months' existence, first at Cape May, N. J., then at Fox Hills, Staten Island. In his candidate's thesis before this same society a year ago, he made this experience the subject of an intimate study. He desires to refer to this work and his conclusions, that we may contrast the benefit to be gained by mechanical means over against the gain from lip reading.

What type of pupil were we dealing with? There were 108 altogether. All but nine were American born. They were young, averaging 26 years of age, and had been of sound body and mind when entering military service. They came chiefly from the so-called laboring class: farmers, laborers, clerks, machinists; and their previous education was low, as but one-fifth had gone beyond a grammar school education, and one-quarter of them were illiterate. The arbitrary qualification for admission to the school was the inability to hear ordinary conversation at five feet. Of the resulting 108 pupils, 32 showed "total deafness," 13 could hear only a "shout," 24 heard more than a "shout" but less than "conversation" at one foot. The extreme types of deafness were auditory nerve forms, while the less severe cases were of catarrhal or suppurative origin. Fifty-two became suddenly deaf, 56 were progressively deafened.

What were the methods employed? Nine of our teachers devoted their entire time to this work. Since these men were

there for the primary purpose of learning lip reading our effort was to give them all they could stand without undue tire. We wanted to saturate them with it. Each pupil had two lessons in the morning of forty-five minutes each and a third in the afternoon of thirty minutes' duration. He had the sole attention of his teacher for the lesson period. She carefully and systematically arranged his work and took him from easy lip sounds to harder tongue and palatal sounds, and then arranged these varying mouth positions and movements in words, phrases, sentences and stories; but the pupil was not conscious of this systematic progression. His sole task was to read his teacher's lips. It was further found best to change the teacher from time to time. This helped him to get used to different speech movements.

What were our early results? Not very good. Failure to see any good to be derived, lack of ambition, fear that learning the art would make them able to support themselves and so relieve the government of having to pension them, despondency, ignorance, laziness, all played their part to create an early inertia which only the persistent zeal, optimism and patience of the teachers could combat. But the inertia had to give way against such pressure.

The big unlettered soldier was being convinced against his will that such practice sentences as "she bought a cap and a map for the baby" must contain profound interest. One man's experience during this stage of transition will illustrate. He started willingly enough, but did not have much stamina and became quickly discouraged. Then he refused to work at class; found every excuse he could to skip attendance. He was referred back to the medical officer for discipline. Neither reasoning nor censure nor the threat of punishment availed. His discharge papers were started and he was taken out of the school. But one of the teachers was not satisfied and gained permission to see what she could do. The bullying, flattery, ragging and entreaty now showered on this luckless man were worthy of a better cause. He sulked, and he yawned; she kept after him. He broke down and cried; she called him a quitter and scolded him. The upshot was that she had that fellow following her around like a trained dog. He worked as though his life depended upon it. His nights were even sleepless from

worry, his eyes became congested from concentrated strain to watch lip movements. A little divine fire had gotten into his soul. He implored his officer to postpone his discharge proceedings, but was told that he had had his chance and now he must take the consequences. So he hurried back to his teacher for more work. The discharge papers came but were pigeonholed until the man had had his full opportunity and the teacher had realized the joy of returning yet another war deafened wreck to useful and happy citizenship. This case is extreme, but it illustrates well many another case of labor and accomplishment and shows how seriously the work was undertaken. The inertia was conquered, enthusiasm followed, optimism prevailed.

All were not hard cases. Many seemed to have an aptitude for lip reading. For most, three weeks of training was sufficient for them to get along fairly well. Some got much delight out of going to dances, where they could see and feel the rhythm of the music and perhaps hear a little. They conversed as entertainingly, the ladies said, as their hearing fellows. When one boy, totally deaf, asked for the day off that he might go gunning for duck in the marshes, we feared that we were taking chances. But he got more birds than anyone else in the party, nor was anyone hurt. Many of the members of our society recall seeing some of the men at the Atlantic City meetings in 1919, and remember how easy it was to talk with them. Some became so proficient that it really seemed they could hear. One man went home on a furlough and it was two days before his people realized his deafness.

We graded them into five classes. Seventy-four per cent finished in the first two gradations, which meant that they could understand over 80 per cent of what their teachers said to them. It is interesting to note that the best lip readers took the shortest time to complete the work. The average time for the entire school was 2.7 months, a remarkable showing when one realizes that this average includes such hard cases as those meningitic, where a definite slowing of cerebration had followed the disease. Our statistics show that the amount of the man's education made practically no difference in his skill as a lip reader, nor did the degree of deafness seem to have a bearing. We were fortunate in being able to follow these

men into civil life through the Federal Board. Reports came back to Washington from all, and it was gratifying to read the enthusiastic reports from the Red Cross and governmental agencies. Some of them wrote that they had to make more than one visit in order to convince themselves that these men did not hear. Many were going into schools of various kinds and a few even into universities. One man hopes to go through Massachusetts Institute of Technology and graduate as an architect; another passed the first year at Worcester Polytechnic Institute and only discontinued because his eyes gave out.

A detailed discussion cannot be entered into here, but I think you will agree with me that the cause of our phenomenal success in the face of such handicaps was not the remarkable work of our specially trained and enthusiastic teachers, and not our military control over the men, but our intensive program. Hard, concentrated effort and continued practice seem essential to the best accomplishment. With such effort our experience shows that all but the mentally deficient can succeed.

This brings us to the third and last phase under discussion: The cooperative work for the deaf now being done in this country through school and guild and association. Tables published last year by the "Annals of the Deaf" show 158 public residential schools for deaf children in the United States, 76 public day schools and 18 denominational and private schools. Canada has 7 schools. In the last number of the "Volta Review" are listed fifty-eight lip reading schools for the adult deaf. The hard of hearing (who for brevity's sake have been classified in this paper as "deaf") have gathered themselves together in associations for mutual betterment and encouragement. There are twenty-four scattered throughout the country. Fifteen of these have become constituent bodies of the "American Federation of Organizations for the Hard of Hearing," of which Dr. Wendell C. Phillips has been president for three years and is now succeeded by Dr. Harold Hays, while Drs. Richardson and Shambaugh have been active in its affairs as directors. Canada boasts two clubs and Scotland one. The New York League has about a thousand members, that in Philadelphia had 803 last June, the Boston Guild 490. Note what the

New York League does where Harold Hays is the leader: Has headquarters occupying one floor of an office building; gives free instruction in lip reading; has a placement and vocational bureau; has industrial, educational and welfare departments, recreation work and nine clubs; and publishes a monthly newspaper. As secretaries or as directors of these guilds, one finds men and women devoting their talents and their lives to the betterment of the deaf in their community. Here the new member, discouraged, perhaps despondent, finds companionship, encouragement, cheer. He learns how and where to secure standard hearing devices, is warned against the charlatan. He joins the lip reading class. He is urged to take active part in the club's affairs. All about him are people who, though deaf, are making their lives count. He realizes he has magnified his loss and that other senses and other attributes can easily make up for this deficiency. Nor is this all. These organizations are becoming an increasing power. Leaders are included in their membership. By local and national organization they are bettering the cause of the deaf throughout our country. Such is the happy fruition of the work begun by Dr. Bell and his colaborers fifty years ago.

Three phases of "aids to hearing" have been discussed. Should emphasis be placed on any one? The conclusion is that the deaf patient should be urgently encouraged to use every helpful agency. He must be reminded that he has lost but one of his senses and that those remaining offer every facility for a life full of joy and service and accomplishment. Through the varied aids at his disposal his lot is becoming increasingly easy. The day is near at hand when he will no longer find himself so handicapped.

19 ELM STREET.

LVI.

MASTOIDITIS IN THE AGED.*

BY RALPH A. FENTON, A. B., M. D.,

PORTLAND, ORE.

Mastoiditis is relatively a rare ailment in people of advanced age. Now and again cases crop up in the literature, while doubtless many more are unreported, or are included in series catalogued by reason of special complications. Consideration of various hospital and infirmary reports during recent years would indicate between 1.5 and 2 per cent of mastoid cases occurring in persons past seventy. Sprague¹ in a series of 200 reported four over seventy years; one was fatal from erysipelas and septicemia.

The insidious character of mastoiditis in the aged is well shown in reported cases. Classed very frequently before 1890 as septicemia, as brain fever or brain abscess, as pyemia, these cases rarely were considered in the light of discoveries made by von Troltsch, Pagenstecher² and others a generation before. Bryant³ reports a woman of 63, with whom he waited thirteen days after the disappearance of mild unilateral mastoid symptoms, avoiding operation because of apyrexia. Death occurred apparently from streptococcus bronchitis, but on postmortem examination extensive double mastoiditis was found, with an intact tympanic membrane on one side.

Butler⁴ cites the case of a man of 84, having acute bronchitis and no previous otitis, admitted in May, 1919, with a temperature of 99, who remained slightly ill for ten days. The bronchitis was due to a mixture of streptococci and staphylococci. On the twelfth day his temperature rose suddenly to 102, without a chill. The left auricle became very tender, and next day there was spontaneous rupture of foul pus into the external auditory meatus. In two days more there was great tenderness over mastoid tip and antrum, and the patient

*Thesis accepted by the American Laryngological, Rhinological and Otological Society.

begged for relief. The mastoid was opened under local anesthesia, with 2 per cent cocain swabbed on and one-fourth per cent injected subperiosteally. Temperature became normal in five days, when a low grade erysipelas supervened, lasting ten days longer. The patient was discharged late in June, 1919, with normal hearing and the wound perfectly healed.

A similar case is reported by Saunders:⁵ A retired physician of 70, with no mastoid tenderness, having pain shooting to the vertex and forehead, was advised on the sixteenth day to be operated, but refused until the forty-sixth day. During a two-hour gas ether operation the entire left mastoid was found excavated, with perisinus and epidural abscesses. There was drainage of cerebrospinal fluid for ten days. Although the prognosis was bad, the ear was healed dry in two months. This man remained well save for a slight attack of right otitis until age 76, when his right ear became involved after a nasopharyngeal infection, Shrapnell's membrane being mainly affected. There was little pain, but on operation, which was not delayed, another perisinus abscess was found. This operation took one hour and forty-five minutes under gas-ether. A pure culture of streptococcus capsulatus was found. The dressing was iodoform gauze for six days, then bismuth-iodoform-petrolatum (BIP) paste; he left the hospital in two weeks, and the wound was healed in three. Five weeks post-operative he had a slight vertigo and was unconscious for a short time, but has since been well.

Certain generalizations will be made from brief reports of three cases in our own service, aged 71, 76 and 82; also certain phases of three others, aged 60, 63 and 64.

Case 1.—L. W., widow, 71, seen January 7, 1920, gave a history of throbbing pain in the left side of the head for five days before. On incision of the bulging pale drum there was a free flow of thin yellow pus. Operation next day (temperature remaining 100 to 97, pain unrelieved) under gas-ether, revealed a large pneumatic mastoid filled with necrotic gray granulations. A streptococcus was present in pure culture. The operating time was twelve minutes, from incision to insertion of stitches; the dressing was dichloramin chlorcosane oil, 2 per cent, with a short gauze plug in the lower angle of the wound and two silkworm stitches above. Low grade ery-

sipelas came on two days postoperative, with temperature to 100.4, and lasted six days. This did not prevent the patient from sitting up in bed and lamenting her appearance. She was out of bed the tenth day, and home with the wound healed on the twentieth day. A short strand of silkworm gut, coiled up for use as a drain, was overlooked in one of the later dressings by the interne and remained encysted until its extrusion eight months later from the lower angle of the wound. Hearing two weeks after the operation and thereafter was one-third normal, better than one-fourth normal rating of the unaffected ear. This patient is still alive and well.

Case 2.—J. K., widow, 76, gave the history of an acute right lobar pneumonia ten days before, following closely upon her husband's death. The right ear had been sore for four days; there was intense pain in the side of the head, but little or no mastoid tenderness. Temperature ranged from 100 to 100.6; white count was 11,000. A small amount of thin streptococcic pus came out after free paracentesis. Operation was avoided for one week more, when the mastoid was opened (April 21, 1921) under gas-oxygen, as pain was increasing and drainage seemed inadequate. The entire process, large and pneumatic, was an abscess cavity; the sinus was exposed for a centimeter, and the necrotic process had proceeded to the digastric fossa. Operative time was eleven minutes; oil dressing; two sutures. This patient's postoperative temperature did not go above 99, and she went home in one week. The wound was healed and normal hearing restored in twenty days; the patient is still in good health.

Case 3.—A. L., widow, 82, was totally deaf and complained that her ears had been very painful for a week past. A doctor had advised her to "let them break." She had not slept for two nights; both mastoids were very tender. The temperature was 103.8, white count 16,000, and albumin and acetone were present in considerable amounts in the urine. Double operation was done February 4, 1922, under gas-oxygen. Very hard cortex was encountered on both sides; the cells were large and filled with very foul greenish pus, mixed streptococcus and staphylococcus. On the left side, as much bleeding occurred from a large emissary entering the exposed sinus, tight roll packing was used. Operating time right side was eleven

minutes; left, fourteen minutes; oil dressing; two stitches. Temperature postoperative ranged from 101 to 99; albumin was gone after one week. This aged and infirm woman left the hospital in eighteen days, and both wounds had healed by the twenty-sixth day. Her former chronic catarrhal deafness was of course unchanged. No inflammatory otitis occurred again. This patient died, several months later, from pulmonary hemorrhage during a slight coughing attack in the course of an acute bronchitis.

In the case of B. J. K., man of 60, cardiovascular patient, an acute right otitis had been permitted to rupture the drum membrane behind an almost occlusive exostosis of the canal. For six weeks, until his pain was almost unendurable, he had received palliative treatment in a distant town. At operation, February 25, 1920, the usual large mastoid cavity was found filled with fetid yellow pus. The cortex was very hard, and there was much deep bleeding; operative time, twenty minutes. This huge excavation filled in but slowly, and the ear was not dry for thirty-six days.

The case of L. H., man of 64, is noteworthy because after a delayed operation (1917), at which a very large cavity was found, and after which the previously high temperature came down to normal, the patient was discovered dead in bed on the morning of the fifth day postoperative. Death was due to coronary embolism; the patient was markedly arteriosclerotic.

The case of E. T., widow of 63, is of interest in that it followed by one week a severe tonsillitis, in a person with chronic bilateral posterior ethmoiditis. The very hard cortex masked an extensive zygomatic cell group. Under gas-oxygen, February 16, 1922, her operation took eleven minutes. She was up on the fifth and home on the eighth day, but has had recurrent mucoid discharge from the ear, following upon certain colds. With treatment of the ethmoiditis the aural discharge has finally ceased.

Conclusions based upon so few reported and observed cases must be drawn with caution. Avoidable errors have been checked in our brief series by the collaboration of internists and laboratory experts, so far as possible.

Sudden lowering of bodily resistance, followed by acute bronchitis or by acute exacerbation of chronic bronchitis, may

be noted as a frequent factor in the etiology of mastoiditis in the aged. Preexistent otitis media, perhaps long forgotten by the patient, must not be ignored in the history, when an estimate of possible danger to the mastoid is made during acute otitis of old people. The remarkable discoveries of Wittmaack⁶ in mastoid pathology, interpreted in America by Norval Pierce, may explain the easy cure of some elderly persons, while others, after recurrent "colds" and "catarrhal" deafness, suffer at last a ready invasion of the abnormally developed mastoid. Arrest of normal pneumatization and ingrowth of abnormal epithelial masses and vascular channels may have existed in such persons since childhood. Also, as suggested by Jones,⁷ these abnormalities and also the flinty cortex and main trabeculae of advanced age may be simulated by the early aging process which accompanies syphilis, lepra and tuberculosis, so that mastoid conditions in luetics of fifty may resemble those normally deferred until three score and ten. Thin minor cellular divisions break down with great speed in the aged, and even the resistant sinus plate or tegmen may display extensive openings, without the warnings of rapid temperature fluctuation, increasing leukocytosis and polymorphonuclear percentage found under similar circumstances in the young and robust. This speedy advance, often with pale membrana tympani, is of course far more likely with the streptococcus mucosus.

Pain in the aged is very likely to be felt elsewhere than in the affected mastoid—referred to the vertex, in front of the ear, down the neck or to the forehead. Tenderness over the tip or antrum may not be marked at any time, owing to the hard cortex. The tympanic membrane may not bulge, may show little or no redness, and that only in Shrapnell's membrane, yet may pour out whitish or yellow pus under pressure when incised. If comparison be made with the sound side, dropping of the posterosuperior wall of the canal will be found not infrequently. Leukocytosis and temperature may not show much increase; such indices of bodily resistance are not raised by the old and enfeebled.

Preliminary radiographs have much confirmatory value regarding the destruction of bony trabeculae in cases otherwise

obscure; but here, as at other ages, the ray must have clinical backing.

If the ordinary conservative measures prove ineffectual and operation becomes imperative, certain points demand respect:

1. Complete preparedness of everything and everybody concerned, before the anesthetic starts; shaving, skin, draperies, aprons, instruments, solutions, sutures, drainage, dressings, adhesive, anesthetist, nurses, surgeon and assistant. This commonplace is nowhere more essential than in dealing with old persons, who tolerate long delay on the operating table very badly. A warm room and a warm and properly padded table are not infrequently forgotten, to the dire discomfort of the patient.

2. Anesthesia should be the easiest possible; preceded by an opiate, usually well borne in combination with atropin; and in general gas-oxygen will be found best, with warmed ether vapor as an adjunct if strictly necessary. Danger from acidosis and renal irritation will forbid ether even more than the respiratory hazards. Local anesthesia, preferably by the technic of Graham,⁸ following Neumann—injection of 2 per cent novocain, paying special attention to the external auditory meatus—may be used in imperative cases. Nevertheless, the psychologic risk from the needle pricks and from the grinding and crunching bone work must not be forgotten. White⁹ reports a case, aged 81, started under local, in which ether had finally to be used.

3. Speed by the operative team means only a shortening of the shock and of the anesthetic risk; it must never be an end in itself. In no class of cases, however, must the surgeon be more certain of his way. Precious minutes ought not to be sacrificed tying surface vessels which an Allport retractor would hold; time must not be frittered away in the search for the antrum, even though old bone be brittle as glass and hard as adamant. Meticulous polishing is unnecessary in the cavities that are usually laid bare in operations on the aged.

4. The wound toilet and dressings must be rapid but thorough. Bone chips, suture ends, gauze frayings, are ill borne in aged structures with poor reparative power. Tight gauze packing is a rather barbarous infliction upon old people. An oily dressing, made with dichloramin—2 per cent—chlorcosane

dropped into the cavity and held there by a short strip of narrow gauze packing folded into the lower angle of the wound, as suggested by Fenton,¹⁰ or injected through small soft rubber tubes in the upper and lower angles of the wound, according to Kistner,¹¹ is by far the most comfortable, as it is the swiftest dressing in the destruction of suppurative processes. Aqueous Carrel-Dakin solution may be similarly injected, as proposed by Mahu,¹² but has the disadvantage of requiring injection every two hours and of maceration of the skin. The latter difficulty may be controlled by vaselin or zinc ointment about the wound. Bacteriologic control of the wound, by daily counting several fields from the wound secretion, will permit suturing as soon as but one organism is found to a field. This refinement is not, however, a necessity with the oil dressings.

Erysipelas may readily occur from tight bandaging, poor circulation, abraded skin or chemical dermatitis. Usually mild, it may condition grave medical complications.

It must never be forgotten that the old person is far more a medical than a surgical risk; and the internist must be constantly on guard against hypostatic pneumonias, pleurisies and the cardiac and renal disorders incident to sudden septic infective processes in those of minimal resistance. Further, the effect of suggestion, in showing the old person as soon as may be that his hearing remains and is improving, is a valuable aid in his convalescence from mastoiditis.

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LVII.

ALCOHOL INJECTIONS AS A POSSIBLE ADJUNCT
TO TONSILLECTOMY UNDER LOCAL
ANESTHESIA.*

BY ROBERT SONNENSCHNEN, M. D.,

CHICAGO.

The writer has always favored the use of a local anesthetic in tonsillectomies wherever it could possibly be used. The advantages of local anesthesia seem to lie in several facts. First, there is either no nausea or, if any, much less than there is with general anesthesia. Secondly, there is the avoidance of aspiration pneumonia. Third, the time of operation is usually considerably shorter than where a general anesthetic is used, particularly in adults. Fourth, there is usually less immediate discomfort in that the patient can take ice, fluids, sedatives, etc., shortly after the operation, whereas with ether it is necessary to deprive the patient of these things for a considerable length of time. Fifth, there is the avoidance of the ill effects that ether or chloroform may have upon the general system, such as cardiac depression, nephritis, or the induction of a coma in diabetes. Sixth, the swallowing of blood and the consequent vomiting are eliminated, in that the patient is able during a local anesthesia to expectorate whatever blood accumulates in the throat.

We have employed local anesthesia in tonsillectomies for many years in all cases above the age of 14 or 15, unless the individual was too apprehensive of the operation or the pharyngeal reflex uncontrollably active. No hypodermic was given before the operation unless the individual needed a sedative. We have often observed that when morphin was used the patient was often markedly nauseated thereby and was likely to vomit while being anesthetized or during the operation.

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Means Employed.—Years ago we injected the mucosa about the tonsil with one-half to one per cent novocain, with or without the addition of adrenalin. During the war novocain was unobtainable at the hospital at which most of our work was done, so we employed apothecin in the same dilution and have ever since used it with great satisfaction in many hundreds of nose and throat operations. The only unfavorable effect noted a few times was a moderate cyanosis or some fainting on the part of the patient. The latter symptom, however, was largely psychic, since it occurred a number of times before the needle had penetrated the tissues. Before infiltrating the mucosa the pharynx was, in some of the patients, swabbed with a small amount of 10 per cent cocain to reduce the pharyngeal reflex and render entrance of the needle less disagreeable. The writer has for many years, when injecting the region of the posterior pillars, entered the needle on the posterior and not the anterior surface. Otherwise the swelling of the posterior pillars causes a distortion of the parts so that it is rather difficult to see where the tonsil ends and the posterior pillar begins; but if the injection is made on the posterior surface of the posterior pillar this does not occur. We do not recall ever having seen this point mentioned in the literature.

In 1909, Yankauer described a most excellent method of producing local anesthesia for tonsillectomy by blocking the posterior palatine nerve at its emergence from the posterior palatine foramen. He states: "The tonsils are supplied by the circumtonsillar plexus derived below from the tonsillar branches of the glossopharyngeal, and above from the middle and posterior palatine nerves. The glossopharyngeal is the smallest of the cranial nerves and its tonsillar branches are small; it is, moreover, a nerve of special rather than of general sensation, and its tonsillar branches may for practical purposes be neglected. The middle and posterior palatine nerves are derived from Meckel's ganglion of the fifth nerve, which is the principal nerve of sensation for the entire head. These nerves leave the sphenopalatine fossa through the accessory posterior palatine foramen, two small openings in the posterior part of the hard palate at the junction of the alveolar process with the horizontal part of the bone. These two nerves supply

not only the tonsil itself but the soft palate, both pillars and a part of the lateral pharyngeal wall. The two openings are very close together, but their location is not marked by any special landmarks on the surface of the mucosa of the mouth, so that the point for injection must be determined by arbitrary measures. There are two methods: First, if the last upper molar tooth is present begin at the posterior end of the margin of the gum of the last molar tooth and measure upward one centimeter—that is, at right angles to the free border of the teeth, and then one centimeter backward. Second, if the last molar tooth is absent, we first determine the location of the tip of the hamular process by placing the finger upon the soft palate; the process can be distinctly felt, as a sharp point of bone.

Then measure one centimeter in an oblique direction upward and forward. When the last molar is present, these two measurements usually meet at the same point; when they do not, an intermediate point is selected. As the mucosa in this region is situated about one centimeter from the bone, the direction in which the needle is inserted is important. A needle with a long shank is used and applied to the mucosa at the point stated, the shank lying across the tongue and in the angle of the mouth on the opposite side. In this position it is inserted until the point touches the bone, when about 1.5 centimeters of a 1 per cent solution of cocain are injected."

The writer tried this method a few times and then for one reason or another discontinued it until the papers of John A. Thompson appeared in 1917 and 1920. He states that "the anterior surface of the tonsils is supplied with sensation by the posterior or small palatine nerve, a branch from the sphenopalatine ganglion. It emerges with a small artery from a separate opening immediately behind the posterior palatine foramen." This lies just anterior to the posterior edge of the hard palate and medially to the last molar tooth. "If the teeth are missing or unerupted, the nerve lies just in front of and internal to the hamular process of the internal pterygoid plate of the sphenoid, which can always be felt through the soft palate. The glossopharyngeal nerve also sends branches to the tonsils." Prof. Knower of the Univervity of Cincinnati

collaborated with Thompson and made sections of the neck as the level of the lower third of the tonsils showing the relation of the glossopharyngeal vessels and the tonsils. The important structures which run through this region are surrounded by a loose connective tissue which is continuous from the pharyngeal wall outward and back in front of the deep prevertebral fascia, to surround the carotids, hypoglossus, superior sympathetic ganglion and plexus, vagus and glossopharyngeal nerves. In blocking nerves it is not necessary to inject the nerve itself, since surface contact is all that is needed. The connective tissue noted by Knower permits us to surround the glossopharyngeal nerve with anesthetic solutions without any danger of injuring the other important structures above mentioned. The injections can be made between the tonsils and anterior pillar, penetrating the constrictor muscles and pharyngeal fascia. A better route is external to the anterior pillar at the junction of the upper and middle thirds of the tonsil. At this point there is only mucous membrane to pierce, and there is no danger of passing the needle through a deep tonsillar crypt as in the first method and of carrying infection into other tissues with serious results. At this level the nerve trunk is three-quarters of an inch from the surface." Thompson and Tucker made a series of experiments to see if undesirable effects would result from surrounding other vessels and nerves of the neck with the anesthetic solution employed (namely, 1 per cent novocain with 10 per cent of adrenalin chlorid solution in the menstruum), after first making a surface application of 20 per cent cocain on the palate and pharynx. Their conclusions were that if the vagus on both sides was surrounded by the anesthetic solution or even a "direct injection of novocain solution was made into the nerve it had no harmful effect." After the preliminary surface application above mentioned Thompson injected 10 minims on each side of the palate and two drams external to each tonsil.

This method was adopted with most excellent results and has usually given us perfect anesthesia and as little bleeding as any other method we have ever attempted. We believe that the operative technic and bleeding are relative matters; whether a sharp instrument, a dull ring or any other appliance is

used, some cases bleed greatly and others very little. This seems to depend on several factors, such as the anatomic peculiarities of the parts, like the presence of rather large veins in unusual places, etc., the direction in which vessels are severed, the clotting time of the individual, the depth in the tissues to which an incision is carried, etc.

Because of its toxicity, the writer has never used cocain, even in weakest concentrations, for submucous injections in the nose or throat. In nasal operations we have applied it topically either in the form of flakes or a 10 or 20 per cent solution together with adrenalin chlorid. In tonsillectomies, as before mentioned, it has been used only by means of swabbing the mucosa of the pillars, palate and posterior pharyngeal walls preliminary to an injection of apothecin or novocain with adrenalin, in very apprehensive individuals or those with very active pharyngeal reflex. However, we have always felt that the less cocain employed the better.

A few weeks ago Dr. Ellison L. Ross read a paper at the April 5, 1923, meeting of the Chicago Laryngological and Otological Society on "Some General Effects of Local Anesthetics Administered as in Tonsillectomy," in which he showed that cocain, even when applied locally, sensitizes the individual to adrenalin so that the blood pressure rises greatly when the latter is injected. This synergistic action produces an increase which is much greater than the combined increase of blood pressure produced by cocain or adrenalin used separately. In animal experiments, this blood pressure increase was shown to be especially high as regards the intracranial venous pressure which increased to 467 per cent. This probably accounts for many of the unpleasant symptoms noted at times when cocain application is followed by the use of adrenalin. Dr. Ross states that "intracranial venous stasis produces cerebral and medullary asphyxia. The former would cause, first, a brief stimulation evidenced by anxiety, restlessness and irritableness. This would be followed by cortical depression which would lead to faintness. Medullary asphyxia would produce the dyspnea, rapid breathing and pallor."

In view of these facts caution should be observed in our anesthesia routine. Dr. Ross stated that if adrenalin be first

used and then a pause of five or ten minutes made until the blood pressure effect has disappeared, the application of cocain will not result in the greatly increased blood pressure noted where cocain is first applied.

Recalling the good results reported by neurologists and surgeons in getting at least temporary relief from pain in cases of neuralgia by injections of alcohol into the nerves or their immediate vicinity, it was deemed worthy of a trial in tonsillectomy. This procedure was contemplated at first not so much for procuring perfect anesthesia during the operation, since we already had that by means of the nerve blocking with apothessin or novocain, as outlined, but it was directed towards reducing or rather mitigating the very painful condition usually present for some days after the operation. Although aided by the American Institute of Medicine, we were unable to find references in the literature on the use of alcohol injections for this purpose, either into the posterior palatine nerve, the glossopharyngeal nerve or the pharynx itself. In order to be certain that the addition of alcohol to a solution containing apothessin and adrenalin would not cause an incompatibility with the production of deleterious substances, the research department of Parke, Davis & Co. was consulted. They assured us that "no chemical antagonism exists between either apothessin or adrenalin and alcohol." They considered it "inadvisable for such a mixture to remain standing more than a few days, since there is always a possibility of slight traces of aldehyde in the alcohol gradually affecting the adrenalin." "In the case of apothessin no indication of instability in the presence of alcohol was found, even after a longer time than a few days." Since we always mix the apothessin, alcohol and adrenalin just before injecting, there can be no danger of any changes occurring in the solution.

Regarding the effect of alcohol on the tissues, Otto May, after quoting Schlosser, Finkelberg and others, concludes that (1) "Alcohol injected near the trunk of a peripheral nerve produces more or less complete necrosis at the point of injection. (2) This change is not ascending; the nerve above the place of injection remaining normal. The cells of origin (ganglion) may show some chromatolysis but no actual per-

manent injury. (3) The conditions produced by the injections are more favorable to regeneration than nerve sections without suture. The anatomic continuity of the nerve trunks favors rapid regeneration, although this may be somewhat retarded by the fibrosis which occurs to a greater or less extent in all alcohol injections." If alcohol was put merely around the nerve its effect, although marked, was transitory; the nerve quickly recovered its function. (Quoted by G. Sluder.) Probably the effect on the other tissues is also a local necrosis followed by a fibrosis.

As to the systemic effects of alcohol, most pharmacologists agree that even in small doses the action on the central nervous system is quite pronounced. It gives rise to a feeling of well being and the individual becomes more self-confident and less shy. Large amounts blunt the sensations. Binz's theory is that alcohol first stimulates the central nervous system and then depresses it. Schmiedeberg's theory is that alcohol depresses the central nervous system from the very beginning, picking out those centers first which were developed last; thus the finer degrees of judgment disappear first. With reference to its action on the circulatory system small doses of alcohol slightly accelerate the pulse and stimulate the heart. The vessels are not much affected, although there is a peripheral vasodilatation. The blood pressure usually rises somewhat.

Various concentrations of alcohol were used by us, from 25 per cent to 95 per cent, but it was found that from 33 to 50 per cent apparently served best in that it had a good effect without the marked reaction. The solution was prepared just before use by mixing about two and one-half drams (10 cc.) of a one-half of one per cent or a one per cent solution of apothesisin with one and one-half drams (6 cc.) of 95 per cent alcohol and adding thereto ten drops of a one to one-thousand adrenalin chlorid solution. In apprehensive persons the mucosa covering the posterior third of the hard palate and the posterior pharyngeal wall was swabbed two or three times with a very small amount of a 10 per cent cocain solution in order to render the entrance of the needle less painful and to diminish the pharyngeal reflex; but where this preliminary cocain application was entirely omitted the anesthesia was just

as good. A straight needle attached to a syringe of one dram (4 cc.) capacity was used. About five drops (0.3 cc.) were injected on each side just lateral to the last upper molar tooth just anterior to the posterior edge of the hard palate. About one or one and a half drams (4 to 6 cc.) were injected external to each tonsil into the areolar tissue at the level of the junction of the upper and middle third of the tonsil and a small amount, six or seven drops (0.5 cc.) on a level with the base of the tonsil. At times a few drops were also injected into the posterior surface of the posterior pillars. After the region of one tonsil was completely injected, the other one was likewise treated and then a pause of from five to eight minutes was made before beginning the removal of the first tonsil. Dissection with knife and snare was done in practically all these operations, which now number 92 private cases and a very considerable number of ward cases.

Results Obtained.—The anesthesia produced by the method outlined was very good. As previously stated in this paper, excellent results, however, had already been obtained by the use of apothecin alone. The advantages which we see from the addition of alcohol to the anesthetic solutions are several. First, there were practically no toxic effects produced from the anesthetic. This may possibly be due to the fact that less anesthetic solution was used than otherwise would have been the case, since one-third or at times even one-half of the fluid used for injection consisted of alcohol. The report of the Committee on Local Anesthesia of the Section on Ear, Nose and Throat of the American Medical Association, on which the writer had the honor of serving, showed that unfavorable symptoms were rarely caused by novocain and apothecin solutions when properly used. It seems reasonable to suppose that the dilution of the anesthetic by the addition of alcohol renders the probability of trouble more remote. Secondly, there was much less syncope. The class of patients which the writer had to operate upon included many very excitable and apprehensive individuals who ordinarily cause a surgeon much trouble in that they become very "nervous" and often "faint" or threaten to do so during the course of the operation itself. Since using the alcohol, however, we have had practically no trouble from this source. Thirdly, there has seemed to be con-

siderably less postoperative bleeding; hemorrhage is, however, a relative matter, and does not seem to depend upon the method of operation but upon other factors previously mentioned. Still, in this series of cases we did notice considerably less postoperative bleeding than usual. This may, however, have been merely coincidental and not consequential. Fourth, during the first 24 to 72 hours most of the patients seemed to have less discomfort than is usually noted. There were some, of course, who complained very bitterly and others who had practically no pain at all. The majority, however, seemed to have much less pain and dysphagia than usual. The committee on local anesthesia recommended that most operations be performed in the reclining or semireclining position, as there was less tendency to syncope, etc., but our cases remained in a sitting posture and there was no difficulty whatever from that standpoint.

While the above mentioned advantages seem to pertain to the use or addition of alcohol to the anesthetic solution, there were several unpleasant features which should be mentioned. The title of this paper gives alcohol as a possible adjunct in local anesthesia for tonsillectomy. It is necessary and important, therefore, to weigh the evidence in order to see whether the advantages far outweigh the disadvantages. Many operations must be performed before it can be definitely stated that alcohol deserves a permanent place in the rôle we have assigned to it. Some of the unpleasant features noted were that there was more pain at the time the fluid was injected than when only novocain or apothésin alone were used, but this pain is transitory. Second, there is always a possibility of some impaired motility of the soft palate for some time, due to a blocking up of the motor nerve. This effect was noted in several patients for a period of from ten to fourteen days, and in one individual there was a complete immobility which persisted for almost four weeks, with recovery after faradization. In the latter case some of the alcoholic anesthetic fluid had been injected to or rather behind the posterior pillar. For this reason we advise (1) omitting injection behind the posterior pillar when employing alcohol; and (2) would also suggest using no greater amount of anesthetic solution for infiltration of the two commonly selected areas (the regions

of the posterior palatine and glossopharyngeal nerves) than is needed to prevent pain. With the impairment of palatal mobility, not only is the speech affected, giving it a so-called nasal twang, but the swallowing of fluids is difficult and they often run out by way of the nose. In a few cases considerable swelling of the tissues was noted for several days, due probably to marked reaction to the alcohol injection. These effects have, however, been observed for some days postoperatively by most men who use infiltration methods of anesthesia, whether dilute cocain, novocain, apothessin or other solutions were employed. In spite of these symptoms, however, we believe that the addition of alcohol to anesthetic solutions deserves a thorough trial, since it apparently has some decided advantages as compared with novocain or apothessin alone.

CONCLUSIONS.

Local anesthesia in tonsillectomies, wherever it can be employed, is preferable to general anesthesia in that it causes less nausea, less vomiting, and less immediate postoperative discomfort. The time of operation is usually shorter than with general anesthetic and there is no danger of aspiration pneumonia. Furthermore, the systemic effects of ether, chloroform, etc., such as nephritis, cardiac depression, the induction of coma in diabetes, etc., are avoided. The addition of alcohol to the anesthetic fluid seems to offer certain advantages in the way of less toxicity, less syncope and less postoperative discomfort and bleeding, which we believe far outweigh the transitory unpleasant symptoms, such as sharp pain at the time of injection and the possibility of temporary interference with the mobility of the palate.

To my associate, Dr. S. J. Pearlman, my thanks are due for much assistance with the operative and other work connected with the preparation of this paper.

Since reading this paper, the author has found that the injection of the anesthetic solution alone (without the addition of alcohol) into the posterior palatine nerve, and injecting as previously described the anesthetic solution containing from 25 to 33 per cent of alcohol into the region of the glossopharynx-

geal nerve, have given all the favorable effects above noted, without causing any interference with the movement of the soft palate after the operation.

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29 EAST MADISON STREET.

LVIII.

THE CLINICAL DIAGNOSIS OF LARYNGEAL TUBERCULOSIS.*

BY FRANK R. SPENCER, A. B., M. D., F. A. C. S.,

BOULDER, COLO.

The purpose of the present paper is to bring before you one of the most frequent complications of tuberculosis, the laryngeal, and to plead for its recognition, with the hope that the realization of the necessity for early diagnosis will lead to the cure of many cases and the arrest of others.

It is hardly necessary for me to state that the type of laryngeal tuberculosis considered is secondary to pulmonary tuberculosis. Such a statement would be superfluous, were it not for the fact that occasionally one hears of the primary form. The primary type is so extremely rare, if indeed it exists at all, that it scarcely deserves serious consideration. Arrow-smith¹ states: "It is conceded today that primary laryngeal tuberculosis probably never occurs and that most individuals who exhibit tuberculous manifestations in mature life have been infected in childhood or infancy, the infection remaining latent as a rule, until some adventitious stimulus spurs it into activity in all of its protean forms." Wood,² however, believes that it is unquestionable that primary tuberculosis of the larynx has occurred, though it is exceedingly rare.

Many laryngologists of wide experience, such as Rr. Robert Levy, state that the primary type of the disease has almost never been encountered in their many years of experience in the diagnosis of laryngeal tuberculosis. Hastings³ says: "Tuberculosis of the larynx is rarely, if ever, primary. The primary focus may be undemonstrable except by autopsy. In view of the numerous positive tuberculin test observations and confirmatory autopsy records in apparently nontuberculous

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patients a diagnosis of primary tuberculosis of the larynx can well be questioned, certainly a verdict of 'not proven,' so far as its being primary is legitimate, until a searching autopsy is made." Levy made a similar statement in discussing Hurd's⁴ paper. Sir St. Clair Thomson⁵ states: "Primary tuberculosis of the larynx, as shown by postmortem integrity of the lungs, is so extremely rare that for practical purposes its possibility may be neglected. When cases are met with where no pulmonary lesion can be detected this is simply because our present methods of investigation are inadequate to detect early or limited deposits in the chest."

Inasmuch as the laryngeal complication is almost as frequent as the intestinal form in patients suffering from pulmonary tuberculosis, this subject deserves serious consideration from the entire medical profession. Because some of us live and practice in Colorado we can appreciate the seriousness of the problem confronting our profession on account of the large number of people who come to the West annually in search of health. That we should and do feel the responsibility more keenly than some others is only natural. Levy⁶ has said: "No subject in medicine possesses a more extensive or interesting history than tuberculosis, nor can any disease boast of more attempts at solving the problem of its treatment with less permanent, definite and satisfactory results." Bane⁷ states that vigorous efforts should be put forth to check the local disease. Dean⁸ and Mullin⁹ have shown the desirability of cooperation between the laryngologist and internist if the best results are to be obtained for the patient.

Brown¹⁰ says that laryngeal tuberculosis occurs in about 25 per cent or more of adults with pulmonary tuberculosis, slightly more in men than in women, and next to tuberculous enteritis and colitis is the most frequent complication of pulmonary tuberculosis, due most likely to direct infection of the part with the sputum. "Even early cases—cases in the incipient or minimal stage—are not spared (12 per cent), but as the pulmonary disease progresses the laryngeal complication becomes more frequent (moderately advanced, 26 per cent; far advanced, 45 per cent). The significance, then, of a complication so frequently seen among patients with pulmonary

tuberculosis cannot be exaggerated." Thomson⁵ gives very similar percentages in his textbook.

One of the very earliest symptoms about which a patient will complain is a slight huskiness of the voice. The laryngologist should be on the alert for the early symptoms. Minor,¹¹ Curtis,¹² Thompson,¹³ Carmody,¹⁴ Grayson¹⁵ and many others have emphasized the importance of an early diagnosis. At first this slight huskiness may not be constant, but is present only upon getting up in the morning or after talking later in the day. As the disease progresses in the larynx the huskiness may become constant and may even amount to decided hoarseness. It has not been our experience that aphonia usually begins early in the disease and persists, except in the severe or rapidly fatal cases.

Some patients have so little involvement of the lungs that the laryngeal symptoms predominate, while others are not aware of any pulmonary disease. In fact, early cases may rarely heal spontaneously. Levy¹⁶ has recently mentioned this. Some of the victims like to believe their cough and expectoration are due entirely to the laryngitis. In this belief patients may be sincere, but in some cases it is because they are hypersensitive mentally regarding their disease and are trying to conceal the pulmonary lesion. It is in just such mild cases as this that a very careful physical examination of the chest by a competent internist, supplemented by stereoscopic X-rays, will show the slight but positive involvement of the lungs even very early. That such early cases should, and frequently do, seek the advice of a laryngologist first is only to be expected. Hence we as laryngologists must be on the alert in the recognition of the earliest laryngeal manifestations clinically. Mullin¹⁷ has urged greater laryngologic responsibility in such cases, because of the importance of an early diagnosis.

Solenberger¹⁸ found "mere weakening of the voice, vocal fatigue on talking, hoarseness, a sense of dryness in the throat, troublesome localized tickling, a lumplike tickling on swallowing, pain, a feeling of languor or debility, and increased dry cough," all important in the early stage of the laryngeal disease. He mentioned also recurrent acute laryngitis as a frequent symptom. He stated: "These signs no doubt often

herald the approach of the disease and should always be an indication for speedy laryngoscopic examination. But they are also signs of actual involvement. Sometimes nearly all these external signs are absent in the process of involvement, even to the point of disintegration."

In moderately severe types the decided hoarseness or even aphonia with cough, expectoration, loss of weight, anemia, anorexia or sometimes dysphagia present an almost unmistakable picture to those experienced in the diagnosis and treatment of laryngeal tuberculosis. It follows, almost without saying, that with aphonia, dysphagia, anorexia, secondary anemia, emaciation, constant cough and expectoration, night sweats, fever, etc., the clinical picture is too evident almost before the larynx is examined. If this deplorable stage can be prevented by an early diagnosis a great deal of suffering and loss of human life can be avoided.

In a paper which the author¹⁹ read before the American Academy of Ophthalmology and Oto-Laryngology in September 1922, the earliest sign of this disease was given as infiltration over the posterior pharyngeal wall. Dr. Robert Levy, in a personal communication, fittingly suggested that it had been his experience that vascular changes often occur earlier than infiltration. I have, therefore, included hyperemia and anemia as the earliest changes in the following classification:

1. Vascular Changes
 - a. Anemia
 - b. Hyperemia.
2. Infiltration
3. Tuberculoma
4. Ulceration
 - a. Acute or Active
 - b. Chronic or Sluggish
 - c. Superficial
 - d. Deep
5. Edema
6. Perichondritis
7. Chondritis
8. Necrosis

Infiltration often is first manifest at the interarytenoid sulcus, or below this point over the posterior laryngeal wall, and is due to sputum remaining in contact with the mucous membrane for hours at a time during sleep if we accept the older theory, advanced by Louis.²⁰ It is difficult to explain the early involvement of the posterior half of the larynx in any other way. Minor²¹ has recently shown many of the early manifestations involving the posterior half of the larynx. Infection through the lymph and blood stream, as advocated by Heinze, is just as probable. If the infiltration becomes more extensive the lateral walls, the arytenoids, the true and false cords, or even the epiglottis may be involved. Thomson⁵ states that different parts of the larynx are usually involved in the following order: "(1) The arytenoids, (2) the interarytenoid region, (3) the vocal cords, (4) the ventricular bands, (5) the epiglottis." Kyle²² says: "The epiglottis is a favorite site for tubercular infiltration, and this organ may assume a simple globular, puffed form, a thickened crescentic shape, or simulate the Turkish turban—the so-called 'turban' epiglottis." He mentions also the importance of the "club shaped" arytenoids when the latter are infiltrated.

Slight elevations above the surface, due to tubercles or tuberculomata, may occur with the infiltration or may follow the infiltrative stage. These may have a smooth exterior, but are usually uneven and resemble a papilloma. In fact, a tuberculoma is frequently mistaken for a papilloma. Trautman²³ has stated that tuberculoma is among the earliest manifestations.

Tuberculous ulcers in the early stages are superficial and have "mouse eaten or mouse nibbled edges." While they may be acute or active they are far more often chronic or sluggish. They are usually covered with a thin layer of sputum or pus and are superficial. Later in the disease the ulcers may be deep, but they do not have the sharply defined "punched out" edges so often seen in lues. The above clinical picture is by no means always so typical, especially when lues and tuberculosis exist together. Carmody¹⁴ states that where there is much scar tissue we may and do frequently have syphilitic infection also. Grunwald²⁴ has emphasized the importance of watching

for lesions in the larynx due to syphilis and tuberculosis. He shows colored plates in his text which illustrate the dual infections. Mixed infections, which are so common in pulmonary tuberculosis, also mask the objective signs and symptoms. Ulcers are common over the vocal cords but rare over the arytenoids. The ventricular bands and epiglottis are often ulcerated too.

Fetterolf²⁵ has classified the ulcerative stages as follows:

(a) Infiltration with superficial ulceration, (b) infiltration and deep ulceration, and (c) infiltration with superficial and deep ulceration. This classification has much in its favor, because we know that frequently the ulcers form on an infiltrated base.

Edema is so often designated as "ashen gray" when seen in laryngeal tuberculosis, and while it usually occurs late, it may be present early. Personally I always feel less optimistic regarding the prognosis when there is extensive edema of the larynx. The mucous membrane covering the edematous areas looks smooth and tense. The blood supply is sooner or later seriously interfered with because of the constriction of the blood and lymph vessels. The landmarks are obliterated by the edema very much as they are in the nose by hyperplastic rhinitis and ethmoiditis. In the larynx it is rarely a true edema, but it narrows the lumen of the larynx. Lockard's²⁶ textbook illustrates this well and his²⁷ more recent article is well worth reading.

Edema may be preceded by perichondritis. However, the edema often hides a perichondritis or chondritis, and it is by no means easy, or sometimes possible, to say in a given case which type of clinical pathology occurred first. It is almost needless to emphasize that while a clinical classification is important, not all cases are of the textbook type. Syphilis may and often does produce a laryngeal picture not at all unlike that of tuberculosis. Thomson⁵ says: "Perichondritis of the thyroid may produce a swelling of the ventricular band, if the inner surface is attacked. The purulent collection makes its way into the larynx. The external surface of the thyroid plate may also be affected. A swelling then takes place externally which is not inflamed and is seldom painful. An incision into it yields pus and leads down to denuded cartilage."

If the patient lives long enough there is apt to result from the edema necrosis of the cartilages of the larynx with severe dysphagia and a fatal termination. The necrosis of the cartilages may take place slowly but is usually rapid. Thus late in the disease the patient is usually emaciated from the prolonged illness, fever and difficulty in swallowing. Lockard²⁸ has shown microscopically that the necrosis of the cartilage of the epiglottis may be due entirely to tubercle bacilli and that it is not necessarily dependent upon a secondary mixed infection.

In conclusion permit me to state that while an early diagnosis does not always mean a complete recovery, we do know that 70 to 80 per cent of our cases of pulmonary tuberculosis are curable if the diagnosis can be made early and proper treatment begun. Thompson¹³ states in speaking of laryngeal tuberculosis that progress is ready to hand in the making of an earlier diagnosis of local infection. A laryngeal complication serves only to lessen a patient's chances for recovery and often prolongs the time required for an ultimate arrest or cure of the disease. Freudenthal²⁹ believes "The prognosis of an established tuberculosis of the upper air passages is better than it used to be years ago." Thomson³⁰ states that "tuberculosis is one of the most common and most deadly scourges of humanity. There is no other which slays so many of our people in the very prime of their career, for it causes one-third of the total mortality during the chief working years of life. It kills 53,000 individuals annually in the small population of England and Wales. It is the greatest cause of disablement in adult life. It leads to more loss to the family and to the nation than any other single disease. It is one of the saddest afflictions."

Freudenthal²⁹ has advocated that "all patients suffering from pulmonary tuberculosis should be advised to undergo a laryngologic examination, not only when the disease is diagnosed or upon their entering a sanatorium, but also at regular intervals, irrespective of their complaints." Mullin¹⁷ has urged such an examination. Certainly it behooves all of us as laryngologists to be on the alert for the early manifestations if we are to succeed in curing tuberculosis.

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PHYSICIANS' BUILDING.

LIX.

THE DIFFERENTIATION OF NEUROLABYRINTH-
ITIS SYPHILITICA AND LABYRINTHITIS
SYPHILITICA TARDA.

BY GEORGE W. MACKENZIE, M. D.,

PHILADELPHIA.

These two forms of syphilis of the hearing apparatus are entirely different affections occurring at widely different stages of the same general disease. They present different pathologic and clinical aspects and prognoses. Furthermore, they may be found eventually to require different forms of treatment, judging from the different results obtainable from the same form of treatment.

As early as 1907 and 1908, while studying the galvanic reactions of a large series of deafmutes, it was observed that the majority of cases of deafness of the so-called nerve type presented, as might be expected, a negative galvanic reaction of one or both branches of the eighth nerve. These observations covered cases of nerve deafness from many different causes. Prominent in the list of causes, particularly among those developing deafness after puberty, was to be noted syphilis, and the majority of these presented negative electrical reactions of the nerve.

The reactibility of the vestibular branch of the eighth nerve was interpreted as negative when neither the positive nor the negative electrode applied to the external auditory canal, with a current strength of 20 m. a., or as much as the patient could tolerate, produced the slightest alteration in an existing nystagmus or caused a nystagmus to appear that did not exist before.

A corroborative test was generally employed, that of opening and closing the current, using first the cathode and then the anode with a current strength of 6 m. a. If closing the cathode or opening the anode produced no momentary arrest of an existing nystagmus to the opposite side nor caused one

to appear to the same side, the vestibular branch of the eighth nerve was considered nonreactive.

The reactivity of the cochlear branch of the eighth nerve was tested after the same manner as the vestibular; however, the reaction when positive is different. The reactivity of the cochlear branch is indicated by a subjective noise, tinnitus; for instance, the cathode produces in the case of a reactive nerve a distinct tinnitus or increase of an existing tinnitus with a current strength of 4 to 6 m. a. When no such reaction is obtainable with 20 m. a., we may conclude that the nerve is nonreactive, nonconductive; in other words, out of function. In this particular type of case it would be superfluous to try the anodal reaction.

Normally with 6 m. a., closing the cathodal current or opening the anodal produces a momentary, but distinct booming sound in the ear of the side to which the electrode is applied. When no such sound is produced it is accepted as evidence of a negative reactivity of the nerve. A stronger current may be tried if one does not feel satisfied with the test made with 6 m. a.,

The reactivity of both cochlear and vestibular branches of the eighth nerve was observed to be negative in the majority of cases of deafness presenting the history and signs of syphilis. Cases were found now and then among the syphilitic group which, in spite of complete deafness and nonreactibility of the vestibular labyrinth to the turning and caloric tests, presented quite normal electrical reactions. This seemed so odd to us at the time that my collaborator, Dr. Alice V. Mackenzie, was prompted to publish these first two cases in the *Wiener Med. Wochenschrift*. These first two cases, by the way, happened in the clinic on the same day within a few minutes of each other. These experiences, which were so contrary to those we were accustomed to seeing, rather upset our previous calculations and made me, at least, feel that, after all, perhaps Barany was right as he practically expressed it in his now memorable monograph "that the galvanic test had no clinical value." At that time we could not, nor did we, attempt to explain these apparently exceptional cases presenting what then seemed to be freak reactions. We were content merely to publish them.

It was several years before the explanation dawned on us. Since 1907 we have seen many cases of both kinds of deafness of syphilitic origin, namely, those with negative and those with positive electrical reactions. The explanation is simple enough now. On looking back, the chief wonder is that it did not occur to us long before. The galvanic test had been serving us faithfully in differentiating *neurolabyrinthitis syphilitica* and *labyrinthitis syphilitica tarda* without our being aware of the fact prior to five years ago, but in a manner definite to our purpose since. What all this has to do with our subject will be unfolded in the course of the paper.

Let us proceed to discuss in their order the points of differentiation made in the opening paragraph of the paper. The first mentioned was that "these two forms of syphilis of the hearing apparatus are entirely different affections occurring at widely different stages of the same general disease."

Neurolabyrinthitis syphilitica, as the name implies, is a syphilitic inflammation of the eighth nerve and the labyrinth. To be more accurate anatomically, the condition could be better labeled *meningoneurolabyrinthitis*, for the neuritis of the eighth nerve is nothing more than the extension of a primary syphilitic meningitis. If there were no meningitis, there would be no neuritis. Acoustic nerve neuritis results from the extension of meningitis and, compared with the other cranial nerves, we find it the most common form of extension. Syphilitic acoustic nerve neuritis is found much oftener than syphilitic optic neuritis. Whether syphilitic meningitis ever occurs without some involvement of the eighth nerve is doubtful. Judging from the accumulated clinical data of Oscar Beck, Willcutt and the writer, one is led to believe that the majority of cases of secondary syphilis reveals evidences of syphilitic meningitis together with involvement of the eighth nerve. Furthermore, the more pronounced the meningitis is, the more pronounced the neuritis. However, the syphilitic infiltration of the eighth nerve is rarely, if ever, quite so pronounced as the infiltration of the meninges. The pathologic findings of Asai, Hofer and O. Mayer show a gradual diminution of the syphilitic infiltration of the nerve from its origin toward its periphery. In some cases, where the patient succumbs to the virus, the infiltration of the nerve barely reaches the

spiral ganglion, in other cases the infiltration reaches to the labyrinthine spaces. The involvement of the labyrinthine spaces has invariably been found less intensive than that of the nerve stem. In short, the meninges, particularly the soft membranes, are found to be involved to the superlative degree, the nerve to the comparative and the labyrinthine spaces barely positive in the average case. The greatest amount of material for microscopically pathologic study of aural syphilis has comprised the temporal bones of syphilitic babes, because they afford the most excellent material for the purpose. None of the pathologic researchers who have studied the temporal bones of stillborn syphilitic babes or those dying within a few hours after birth have yet been able to find one in which the eighth nerve has escaped involvement. This shows as plainly as can be the vulnerability of the eighth nerve to the virus of syphilis and corroborates completely the clinical findings of the gentlemen mentioned earlier. The hereditary form of syphilis that is fatal to infants before or shortly after birth is typically generalized syphilis, showing all the characteristics of a virulent secondary syphilis of the acquired form.

I have referred to the second form of aural syphilis as labyrinthitis syphilitica tarda to distinguish it from neurolabyrinthitis syphilitica. The object of using these two different terminologies is for the purpose of contrasting the two conditions from the pathologicoanatomic standpoint. In this second form of aural syphilis the labyrinth is primarily involved, and from every indication available at this time the nerve escapes entirely. In the first mentioned condition the nerve is definitely involved and the labyrinth but slightly. In the second, the labyrinth is definitely involved and the nerve apparently not at all. It is clear then that these two conditions, in spite of the fact that they both have a common origin, are "entirely different affections."

Contrasting these two conditions further, we find neurolabyrinthitis occurring during the secondary stage of syphilis, when the virus is widely disseminated throughout the body, a matter of a few weeks or months following infection. It, therefore, occurs early. On the other hand, labyrinthitis syphilitica tarda occurs very late, usually in the hereditary form of syphilis, when it forms one of the symptoms of the complex

known as Hutchinson's triad. It may occur also very late in the acquired form, but it is rather an unusual manifestation of acquired syphilis, while in the hereditary form it may be considered fairly frequent. According to Hutchinson, it never occurs before the fifth year, more often at about the age of puberty, and is rarely, if ever, seen after the thirtieth year. Notwithstanding, the writer saw and reported a case a few years ago of labyrinthitis syphilitica tarda in a congenital syphilitic that had begun when the patient was forty-three years of age. We see, therefore, that these conditions are not only "entirely different affections," from the pathologicoanatomic viewpoint, but they also occur at "widely different stages of the same general disease."

These two conditions "present different pathologic" aspects, as already pointed out. They may be compared with two separate syphilitic conditions; for instance, optic neuritis or neuroretinitis of secondary syphilis and interstitial keratitis, which occurs only very late in syphilis, more often in the hereditary form. Both of these eye conditions can lead to blindness, and both are due to the "same general disease," but in no respect may they be considered as the same pathologicoanatomic entity. Both neurolabyrinthitis syphilitica and neuroretinitis syphilitica occur early in syphilis and occasionally nearly or quite simultaneously. On the other hand, labyrinthitis syphilitica tarda and interstitial keratitis occur very late and often together or shortly following one another. Other points of comparison existing between neurolabyrinthitis syphilitica and neuroretinitis syphilitica appear farther on in the paper as well, too, as the comparison between labyrinthitis syphilitica tarda and interstitial keratitis syphilitica.

The clinical aspects of neurolabyrinthitis syphilitica and labyrinthitis syphilitica tarda differ sufficiently to permit of a ready differentiation.

In the case of neuritis of the eighth nerve, if the involvement is sufficient to impair the normal conducting power of the nerve, the impairment can be detected by the galvanic test conducted after the manner already described. In the case of labyrinthitis, on the other hand, with an intact nerve, the galvanic reaction remains quite normal. The writer has observed many cases of bilateral labyrinthitis syphilitica tarda

with complete loss of vestibular function to the turning and the caloric tests, manifesting perfectly normal reactions to galvanism. A classical case of the kind was shown and reported with minute details a little more than a year ago, before the Philadelphia Laryngological Society, by two students of the Graduate School in Philadelphia. Many cases of the kind have been seen by the writer, both before and since that time. Again, cases of complete bilateral deafness have been seen in which the galvanic reaction of the cochlear branch of the eighth nerve has remained normal. Other cases of bilateral loss, both of the hearing and the vestibular functions, from labyrinthitis syphilitica tarda have been examined with perfectly normal galvanic reactions of both acoustic and vestibular branches of the eighth nerve; so that from the standpoint of clinical diagnosis it is not at all a difficult task for the examiner to distinguish these two processes.

In many other respects these two forms of aural syphilis resemble one another. Both are due to the same general disease, hence the history and findings of syphilis would not be sufficient to solve the question. If, however, the case is one of hereditary syphilis, there is a very strong suspicion that the resulting deafness and impairment of vestibular function are due to labyrinthitis syphilitica tarda and not to neurolabyrinthitis syphilitica. If the case can be proved to be one of acquired syphilis, the suspicion rests more strongly on neurolabyrinthitis.

Cases are met with now and then in which the history and findings point strongly to generalized secondary syphilis but not strongly enough to be absolutely dependable. In this type of case the character of the galvanic findings, together with the functional hearing tests, even in the absence of symptoms referable to the parts, has frequently led to the positive diagnosis of syphilis, to be confirmed later by that most dependable of all, the therapeutic test. The experience of the writer prompts him at this time to the decision that if he were allowed the privilege of selecting but one of two alternatives in arriving at a diagnosis of syphilis, he would unhesitatingly decide in favor of the functional tests in preference to the Wassermann. Again, there are cases of only moderate impairment of function, vestibular or cochlear, and cases of begin-

ning impairment, in which the galvanic and functional hearing tests have given advance information, permitting thereby the institution of treatment earlier than might have been possible otherwise.

In *neuro labyrinthitis* we have, as a rule, bilateral impairment of hearing, which is progressive from the start, but it rarely, if ever, goes on to complete deafness. In *labyrinthitis syphilitica tarda* the onset is more abrupt (apoplectiform in some cases) than in *neuro labyrinthitis* and frequently goes on to complete deafness.

Neuro labyrinthitis syphilitica presents a rather favorable prognosis in those cases in which treatment is begun early and is vigorously pushed. *Labyrinthitis syphilitica tarda*, on the contrary, presents a less favorable prognosis, even when the treatment is begun within a few days of the onset. In the latter condition there is frequently a pronounced improvement following the initiation of treatment, but there is a tendency to relapse in spite of the treatment. Such relapses are common in *labyrinthitis*; exceptional in *neuro labyrinthitis*. Why this is, the writer is unable to answer. But it suggests to us that the same form of treatment is not as definitely indicated in both of these conditions any more so than the same antiluetic treatment will answer equally well for deep interstitial keratitis and *neuroretinitis syphilitica*. In *neuro labyrinthitis syphilitica* and *neuroretinitis syphilitica* antiluetic treatment, particularly mercury, accomplishes apparently wonderful results. On the other hand, in *labyrinthitis syphilitica tarda* and in deep interstitial keratitis this same kind of treatment occasionally works with some satisfaction, but more often it does not, while a few cases are apparently aggravated by the treatment. It is common knowledge on the part of ophthalmologists that the second eye frequently becomes involved with interstitial keratitis while the first is still under treatment. The results of treatment in *labyrinthitis syphilitica tarda* are quite as unsatisfactory as they are in interstitial keratitis. It is possible that the best form of treatment for these last mentioned conditions has not yet been found. It is evident from the foregoing remarks that the two forms of aural syphilis referred to in the paper present not only "different pathologic and clinical aspects and prognoses," but in addition "they

may eventually be found to require different forms of treatment, judging from the different results obtainable from the same form of treatment."

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LX.

PAROTID FISTULÆ AFTER MASTOID OPERATION.

BY W. E. GROVE, M. D., F. A. C. S.,

MILWAUKEE.

The appearance of parotid fistulæ after mastoid operations has been reported several times in the literature, but inasmuch as these reported cases have been very few, it was thought of interest to bring this subject to the attention of this society.

In 1906, when Kretschman reported his first case, he called attention to the absence of any notations concerning them in the literature. He reported two cases. One was a young man of 25, in whom an extensive resection of the entire mastoid process was necessitated. There was a postauricular incision 12 c. m. long, which granulated normally with the usual amount of wound secretion. The main portion of this wound healed in normal time, but he noticed one point at which there was a moon shaped opening, from which a clear watery yellow secretion exuded. This opening was at about the level of the mastoid tip. A sound introduced into it ran about 2 c. m. toward the inferior canal wall.

His second case was a child of 10. The entire mastoid wound healed promptly in eight weeks. Three and a half years later this patient came back, stating that six months before, following a blow on this ear, the wound became moist and had so persisted. In the postauricular scar at the level of the mastoid tip there was a fistula from which a thin limpid fluid exuded during the act of chewing. A sound introduced into it passed about 2 c. m. toward the inferior canal wall.

Combier reported three cases which occurred one and one-half, three and four months after the cure of the mastoid wound. They were all found at the lowest portion of the mastoid wound. Probes could always be passed about 2 c. m. toward the inferior canal wall.

Binnerts reports a case in a child of 5, where a bilateral mastoid operation had been done five months before by another surgeon. Whenever the child chewed or moved the

jaw a fluid appeared from a fistula situated in the lower part of the right scar. There was no similar trouble on the left side.

In addition to these cases, two more were reported by Claus, three from Passow's clinic, and one by Marres of Amsterdam. Cases were also reported by F. C. Schreiber, Philip Hammond and C. Canesteo.

CASE REPORT.

On March 3, 1920, there was admitted to Columbia Hospital a child, aged 8, with an acute mastoid on the right side. This followed an attack of influenza two weeks before. There had been a severe earache three days before, when a paracentesis was done. Following this there was a copious discharge of pus from the ear.

Past history: Earache with every attack of sore throat. Has had several previous attacks of otitis media. Tonsils and adenoids were removed December, 1917. Remainder of the past history negative except for an attack of pneumonia at 18 months.

Physical examination: Brown purulent discharge from right external canal. Tenderness over right mastoid region but no edema. Glands of right side of neck are slightly enlarged. Left ear negative. Chest negative. Abdomen negative. Reflexes all present, but those of extremities are exaggerated. On admission, temperature 103. Pulse 102. Respirations, 22. Blood examination, 10,850 W. B. C. Differential count shows S. M. 4 per cent; L. M., 2 per cent; polys., 85 per cent; eosin., 2 per cent; trans., 6 per cent; baso., 1 per cent.

Operation, March 4, 1920: Typical simple mastoid operation done with a straight vertical postauricular incision extending from the level of the upper attachment of the auricle to the mastoid tip. The cortex was extremely thin. When this was removed extremely large mastoid cells were encountered, which occupied the entire mastoid process down to the extreme tip, and which were everywhere filled with a thin straw colored fluid. Culture from the wound gave Gram + coccus. Blood culture was negative. Wound was packed with an iodoform drain.

March 8, 1920. Marked nasal discharge for last few days.

March 12, 1920. Right dacryocystitis appearing with severe inflammatory signs.

March 16, 1920. Under gas anesthesia the abscess of the right lacrimal sac was incised and a small drain inserted.

March 28, 1920. Patient left hospital with both mastoid and lacrimal wounds in good condition.

May 4, 1920. Patient entered Mt. Sinai Hospital because of a rise of temperature, which had begun one week before. At this time the drum, which had closed, was reincised. There was not much discharge after the paracentesis, but there was swelling and tenderness in the region around the mastoid wound. Under gas anesthesia the mastoid wound was cleaned out and some infected cells in the zygomatic region not reached by the original operation were cleaned out. The elevated temperature fell to normal in three to four days, and the patient left the hospital on May 16, 1920. Between May 26 and May 31 she again reentered the hospital for observation because of a rise of temperature, but this gradually subsided.

The mastoid wound now gradually filled in until it was closed and the patient left town. While away she noticed that the wound became moist whenever she chewed or yawned. When she returned to my observation I made out a small fistula in the lower portion of the mastoid wound leading in the direction of the inferior portion of the auditory canal, and from which a thin limpid secretion was seen to issue whenever she chewed. Various attempts to close this by means of fused silver nitrate but with no success.

In February, 1922, during an attack of scarlet fever an extensive mastoid operation on the left side was necessary. The same very pneumatic mastoid was encountered as upon the right side. The entire mastoid consisted of very large cells extending to the extreme tip, into the zygomatic region and back into the diploic spaces. These cells were all involved in the process and were cleaned out. The lateral sinus was laid bare throughout a large extent because of a perisinus infection but was not opened. During this illness the right mastoid wound broke down, following an acute otitis media, and it was again opened and drained. The mastoid wounds both filled in very slowly because of the lowered resistance of the patient. After they had closed entirely a very tiny fis-

tula appeared at the lower portion of the left mastoid wound similar to that which had occurred two years previously on the other side. As far as I know, this is the only reported case of a bilateral parotid fistula following mastoid operations.

I have not been able to close the fistulae in his case by silver cautery, but as time passes they are gradually closing themselves down so that at the present time very little fluid escapes from them. I have advised resection of the fistula and suture, but this has been refused by the patient.

All of these parotid fistulae have occurred either during the closing of the mastoid wound or very shortly thereafter, with the exception of one of Kretschman's cases, wherein the fistula occurred three and one-half years after the operation. Binnerts believes that they are due to an injury to the parotid gland by a sharp instrument during the operation, but inasmuch as they occur so rarely there must also be some other cooperating factors, such as abnormally large parotid glands extending backward over the mastoid process. This may occasionally occur, according to Graupner, who also found accessory parotid glands on the surface of the mastoid. Another cooperating factor, according to Binnerts, is the curved incision affected by some surgeons for cosmetic reasons. Kretschman also calls attention to the fact that previous infections or inflammations in the parotid may increase its size and push it back into the operative field of a mastoid, where it could then be injured by a separation of the attachment fibers of the sternocleidomastoid muscle.

Another factor, which has occurred to me, is that the gland may enlarge because of infection in its neighborhood at the time of the mastoid itself.

In those fistulae which develop late Combier is of the opinion that the actual damage to the gland is done at the time of operation; that this area then apparently heals with the formation of a retention cyst, which later breaks down, due to some insult or infection.

As to the treatment of these parotid fistulae, Binnerts cauterized the fistula in his case with chromic acid but without success. Kretschman cauterized frequently with fused silver and cured both of his patients after having failed by curetting and cauterizing with the galvanic cautery. Passow was not

able to cure his patients in this manner. He then excised the fistula and sutured the edges. Combier cured his patients by injecting a few drops of tincture of iodine into the sinus. Hammond also excised the fistula and sutured. We must not forget that these sinuses may also close spontaneously.

This report would be incomplete without calling attention to a paper by Kretchman, published in 1914, in which he calls attention to the occurrence of parotid fistulae in the external auditory canal and reports one such case of his own and one of Politzer. Inasmuch as the external canal and the parotid gland lie in such intimate connection it is conceivable that furuncles of the former and abscesses of the latter may form a fistulous connection between them.

In his own case, after cleansing the external canal, he discovered a bleb about 4 m. m. anterior to the drum, and at the juncture of the upper and anterior canal walls, which became more prominent on chewing. An examination of the secretion obtained from this region was pronounced by Abderhalben to be saliva. Politzer's case was similar, except that the fistula was at the juncture of the anterior and inferior canal walls.

Kretchman was of the opinion that there may have been some defects in the formation of the external canal, in which defects some parotid tissue came into close connection with the external canal wall. In both of these cases the middle ear and the sound perceiving apparatus were normal.

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LXI.

LYMPHOID TISSUE IN THE TONSILLAR FOSSÆ FOLLOWING TONSILLECTOMY.

BY B. B. GRICHTER, M. D., AND S. J. PEARLMAN, M. D.,

CHICAGO.

The recurrence of lymphoid tissue in the tonsillar fossæ following apparently complete tonsillectomy is of perennial interest to the surgeon. Pierce¹ divides these recurrences in two groups; one in which the lymphoid tissue at the base of the tongue is drawn up by a contraction of scar tissue in the capsule, and another in which there is a globular mass unconnected with any kind of tissue at the base of the tongue. The appearance of the last named type of lymphoid reformations in tonsillar fossæ, following apparently complete tonsillectomy by competent operators, can, we believe, be explained by a consideration of the development and anatomy of the tonsillar capsule, plicæ and fossæ. This explanation is confirmed by histologic findings in tissue removed from the plicæ and supratonsillar fossæ following tonsillectomy.

The tonsil itself originates in the third month of embryologic development from an eminence, the tonsillar tubercle (Hammar²) in a recess formed from the lower portion of the second pharyngeal pouch. The walls of this recess originate from the second and third visceral arches, which become respectively the anterior and posterior pillars.

The plica triangularis or anterior tonsillar fold springs from the entire length of the anterior pillar and tends to cover the tonsillar recess or sinus like a lid. From the posterior pillar a similar fold, the posterior tonsillar plica, still further narrows the entrance to the sinus. This last structure is, however, not constant.

From the mucosa of the sinus solid epithelial buds or sprouts invade the underlying fibrous tissue. This takes place about the fifth month of embryonal life. These buds may be single or branched. The central cells degenerate and are expelled

as horny plugs, leaving blind crypts which become compressed by the simultaneous development of lymphoid tissue.

The latter originates in the more superficial portions of the tunica propria of the mucosa. The deeper layer of the tunica propria is pushed outward and becomes the capsule. "The capsule covers all surfaces of the tonsil not covered by epithelium. Theoretically it includes only that part of the deeper layer of the fibrous mucosa of the sinus that covers the attached surface of the main bud of the tonsil. As the mucosa of the more internal surfaces of the sinus, including the plicæ, have lymphoid nodules developed in them, often directly continuous at the root of the tonsil with the lymphoid tissue of the main mass, it seems best to consider the capsule as including the fibrous mucosa of the entire sinus, even to the internal rim of the plicæ." (Barnes.³)

"As the lymphoid mass grows it fills more and more completely the tonsillar recess and "finally bursts through the faucial opening of the sinus, the walls of which surround it, much as the calyx surrounds the growing bud. The plicæ, partly because it may be pushed forward by the tonsil and partly because of the forward growth of the tongue, may undergo a greater or less reduction at its inferior insertion. If no reduction takes place the tonsil is suspended as by a sling by the plicæ, and a deep fossa exists between the two anteriorly and inferiorly. Between this condition and one in which only a slight fringe of plica remains anteriorly all gradations may be seen. If a plicæ retrotonsillaris is present, there is a posterior fossa between it and the tonsil which is continuous with the inferior fossa if one exists. The superior lobe of the tonsil expands upward into the cul de sac which forms the superior angle of the sinus, the walls of which surround it like a hood. Between the two an extensive fossa remains which may be quite open or its epithelial surface may be in apposition. This is the supratonsillar fossa. Its size varies with the size of the original pocket and also with the height on the outer wall of the sinus to which the root of the tonsil extends. Its faucial opening is more or less covered by the upper part of the plica so that internally it is continuous with the anterior fossa and, if one exist, with the posterior fossa. The ventral walls of the sinus surrounding the

tonsil have a small amount of lymphoid tissue developed in them, sometimes with shallow crypt formation. This is especially true of the supratonsillar fossa and of the plica anteriorly. This lymphoid tissue is continuous at the tonsillar root with that of the main bud and must be considered as forming part of it. At times the amount of lymphoid tissue developed in the deeper layer of the plica is so great as to form a thick wedge shaped mass, which is continuous with the main bud of the tonsil at its base. Under these conditions the plica has the appearance of being directly attached to the tonsil, and the anterior fossa is converted into a deep vertical crypt. This is the so-called attached plica (Fetterolf).” (Barnes.⁴)

The tissue studied in this work was obtained from children aged from four to fourteen, at the Michael Reese Tonsil and Adenoid Clinic. The tonsils were of varying degrees of hyperplasia, but with no evidence of recent acute inflammation. They were removed under general anesthesia with the Beck-Mueller tonsillotome. This instrument is the one in general and favorite use at this clinic and, as will be shown later, bears no relation to the recurrences. The excised tonsil was first carefully examined and the presence of the intact capsule noted before a specimen was taken from its fossa. Specimens were not taken unless the tonsil was considered successfully removed in one maneuver. Tissue was removed by means of a small punch forceps as near as possible to the anterior pillar where it forms an arch with the posterior pillar; better seen when the former is put on the stretch. No tissue was removed from the region of the lingual tonsil.

Altogether 100 operated fossæ were studied. Serial sections were made and every tenth section was analyzed. In fifteen instances (15 per cent) lymphoid tissue with distinct follicle formation was observed. In some cases shallow crypt formation was present. In ten additional specimens (10 per cent) lymphoid tissue of a diffuse variety was seen. Much of this was microscopic in character. In all, 25 per cent of operated tonsillar cavities exhibited lymphoid tissue. This is probably a minimum figure, because at times only one piece of tissue was removed. Had serial sections been studied, lymphoid tissue would probably be found in a larger number of instances.

We do not conclude that this extratonsillar but intracapsular lymphoid tissue is always responsible for these recurrences. This tissue, however, seems to be the only one from which such recurrences can take place and best explains the reformation of lymphoid masses high up in the fossa which occur now and then at the hands of competent surgeons using the best possible technic. It is true that demonstrable recurrence may not occur in all instances in which lymphoid tissue can be shown to be microscopically present, since factors as yet unknown and indefinable may determine recurrence in one instance and prevent it in another.

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25 E. WASHINGTON ST.
29 E. MADISON ST.

LXII.

CLINICAL AND PATHOLOGIC STUDY OF TONSIL CASES.

BY AUSTIN G. MORRIS, M. D.,

ROCHESTER, N. Y.

The general impression among laryngologists relative to tonsil surgery has changed markedly during the past few years and has assumed a more rational working basis upon which to draw accurate conclusions.

It will be remembered that for many years the custom and practice was to remove obstruction, whether found in the throat or nose. If found in the throat a slicing operation was performed for the removal of the projecting part, and in the nose the removal was made of whatever structure presented the least difficulty. Should the trouble be found in the ears the patient was sure to be subjected to the old reliable treatment, namely, inflation. The result of some of this work has, as might be assumed, been most unsatisfactory. It has in a general way made the patient, treated in past years, a permanent patient. It not only failed to give relief, or at the most only temporarily so, but by the surgical work being misdirected made sufferers who were constantly exploring new fields in hopes of gaining relief. To our own leaders and those abroad we owe a keen sense of gratitude for our larger conception and a basis for reasoning whereby we are able with our present adjuncts to diagnose and by surgery to give relief and comfort.

At the present time the most frequent operation in all ear, nose and throat surgery is the removal of tonsils and adenoids, and justly so, for there is no question that more pathology is found in these structures than in any other in our field. Not only is pathology found in these structures, but, by its presence in them, infection is transmitted to other structures and causes general systemic trouble or perchance local trouble distant from the original focus. Much has been written on focal infection, and I have no desire to add to the voluminous

literature on the subject. I have had glaring failures and brilliant successes, as many others have had.

An experience of something over 10,000 tonsillectomies that I have done because of clinical findings convinces me not only of the reliability of clinical findings as a basis for tonsillectomy, but has also impressed me with the great responsibility that the surgeon should shoulder in this so commonly performed operation of tonsillectomy, namely, the responsibility not only of leaving a good throat but of removing every vestige of infected tissue.

Impressed with the serious infection in many throats presenting themselves for relief, not a few of which had been previously operated upon by some one or other of the incomplete methods, I have had many tonsils, removed during the past four years, subjected to careful histologic and bacteriologic examination.

It is the purpose of this paper to report a resumé of the findings in this series of cases and to add a word or two relative to impressions gained therefrom.

It might be of interest to state, in a preliminary way, something of the method employed in the pathologic examination. The tonsils, immediately after extirpation, were put into sterile gauze by the surgical nurse and taken to the laboratory, where they were fixed in 10 per cent neutral formol. Adenoids were similarly treated. The faucial tonsils were split longitudinally into two equal parts, the sections being taken from the cut face of one part. The other part was retained for possible further study. The ordinary methods of paraffin infiltration and embedding were employed, and the tissues were stained in hematoxylin and eosin. Usually only one section from each tonsil was examined microscopically, although in certain doubtful cases it was found necessary to section all of both tonsils at intervals of about 2 mm.

The exact number of cases so examined was 2321. Aside from the ordinary pyogenic infection, present to a greater or less extent in all tonsils, the most common pathologic condition found was tuberculosis, involving usually either one or both tonsils. In addition, syphilis was demonstrated in one case where spirochetæ were found in large numbers. There were two cases of rather rare mycotic pharyngitis, which involved

the tonsils, and one case of tumor, namely, perivascular endothelioma, in an elderly person, which had metastasized to lymph glands.

As regards the histopathology of tonsils showing tuberculous infection, they fell for the most part into two main groups, although there were a very few cases of a third group, which might even be considered as a subgroup of one of the main divisions. The first group was characterized by the location of tubercles about a crypt or its ramifications. Sometimes they appeared at the base of the crypt near the capsule, while at other times they were more superficially located, either about the mouth of a crypt or beneath the mucosa of the tonsil itself. In one instance the tubercle was traced by serial sections directly into a crypt, which itself was somewhat cystlike from obstruction. Usually this type of tubercle was epithelioid, sometimes containing giant cells, at other times not. Precisely the same type of lesion was at times present in the adenoids, either in conjunction with the tonsils or exclusively in the former. In five cases tuberculosis was found exclusively in adenoids. Practically never was there any considerable amount of caseation in these tubercles and usually none. At times these tubercles appear to coalesce near the surface of the tonsil, with more or less caseation, resulting in erosion and producing tubercular ulcer. This type of case constituted a subdivision of the so-called cryptic type. The cryptic type of case is usually unilateral.

The other main group of cases is characterized by a rather diffuse dissemination of tubercles throughout the tonsil. They do not respect the germ centers, invasion of which structure has not been observed with the cryptic type. Giant cells are more common, and caseation and secondary infections are very frequent. In this group usually both the tonsils and sometimes the adenoids are involved in the manner stated.

It is highly probable that the latter group of cases is secondarily infected, and is but a manifestation of an active tuberculosis elsewhere in the body. On the other hand, it is more probable that the cryptic group represents a primary infection of the tonsil, either by inspiration or ingestion, or perhaps an infection from open pulmonary lesion. Theoretically it is impossible to rule out the presence of tuberculosis elsewhere

in the body, nevertheless, the relation of these tubercles to the crypts themselves, coupled with the usual absence of clinical signs and with the good follow up histories that most of these patients present, makes it highly probable that such cases have not been infected from an open lesion but rather from inspiration or ingestion. It is very probable that some cases of disseminated tuberculosis may have their crypts infected from an open pulmonary lesion resulting in a mixture of the two types of cases. The clinical significance, however, of such cases would be the same as of the disseminated type.

That such pathologic grouping of the cases may have clinical significance has also been suggested by others, in which event such a pathologic report should be of value both to the clinician and to the patient. The cryptic type represents a relative benign infection, as shown by its relative freedom from caseation and secondary infection as well as by its superficial character. With proper watching such cases promised to have no further involvement. With the other group, to be sure, the clinical significance is different. If evidence of active tuberculosis has not been discovered before, such evidence should afford most helpful information for both clinician and the patient. The significance would, of course, be that of any case of active tuberculosis.

Among the rarer conditions of interest were two cases of mycotic pharyngitis involving the tonsils. Clinically, the appearance of the throat was characterized by discrete, raised, yellowish nodules, which were so adherent as to make removal impossible except by the use of a sharp instrument. Section of these nodules disclosed the fact that a hyperkeratosis was present. This process began at the lower layer of the mucosa, which fact explained the tenacity with which the nodules adhered to it. They presented a lamellated arrangement, and cellular elements were few or absent. Stained by Gram-Weigert, threadlike bacteria were seen between the lamellæ, which corresponded well with direct examination of the exudate and macerated nodules. Long, rather thick threadlike organisms were present in addition to many spirochetes and fusiform bacilli. Cultures were not definitive.

The single case in which syphilis could be definitely proved was one in which the tonsils were enormously enlarged and in

which the condition had not been suspected. The cervical lymphatics were quite palpable, but the patient gave persistently a negative history. Histologically, there were present, particularly in the deeper parts of both tonsils, areas of epithelioid tissue, which were vascular, in contrast to that ordinarily seen in tuberculosis. No giant cells were observed. The Levaditi stain disclosed large numbers of typical spirocheta pallida in these areas and in the lymph vessels, as well as around the blood vessels.

The single case in which a tumor was found occurred in an elderly woman with enlarged lymph nodes. Expiration of both tonsils and nodes disclosed the very rare tumor known as a perivascular endothelioma. Its structure was characterized by the presence of dilated lymph channels lined by polyhedral cells with lymphocytes surrounding the small arterioles, as is the case with a true perithelioma.

Tuberculosis was found in 39 of the 2,321 examinations (1.7 per cent), which is usually lower than in groups studied by others, as follows:

Author		T. B.	Per Cent
Albrechts	1,060	3	0.3
Barnes	250	3	2.0
Crowe, Watkins & Rothholz.....	1,000	46	4.6
Judd	1,000	23	2.3
Lewin	200	9	4.5
Robertson	232	19 (?)	8.0
Sewall	772	30	3.9
Willis	108	9	8.0
Weller ¹	8,697	204	2.35

Virchow, in 1864, stated that tuberculosis of the tonsils had not been observed. Cornil, in 1875, is reported to have found ulcerative lesions of the pharynx and tonsils in an otherwise healthy individual. Deplous, in 1878, found tuberculous ulceration of the tonsils in a child with marked pulmonary tuberculosis. Orth experimentally produced tonsil tuberculosis in dogs by feeding them tuberculous tissue. In 1887, Orth reported well authenticated cases of pulmonary tuberculosis in tonsils of children dying from diphtheria, without evidence of tuberculosis elsewhere in the body.

Tuberculosis of the tonsils has been carefully studied and reported in its many phases by Pluder and Fischer,² McBride and Turner,³ Brieger,⁴ Piffel,⁵ Rethi,⁶ and Hynitzsche.⁷ Tonsil tuberculosis occurring during childhood has been investigated by Latham,⁸ Friedmann,¹⁹ Ito,⁹ Kingsford¹⁰ and Mitchell.¹¹

The tonsil as a point of entrance of ingestion; the nature of the infecting organism, whether bovine or human; its relationship to lymphoid hyperplasia and relationship to cervical gland and pulmonary tuberculosis has been dealt with in articles by Gottstein,¹² Grober,¹³ Bandelier,¹⁴ Carmichael,¹⁵ Sewall,¹⁶ Mitchell¹⁷ and Crowe, Watkins and Rothholz.¹⁸

In view of the known effects of tuberculosis or other serious infections of the tonsil, and the incidence of such infection as developed by this and similar studies, the conviction is forced upon one that the question of whether or not to operate in a given case should merit a careful and systematic study of every case presented for examination. In my own experience the method of arriving at such a decision has been a careful noting and recording of the following:

1. History.

2. Clinical examination.

In obtaining the history I have endeavored to obtain the salient facts by the following series of questions:

1. Age.—This seems at first to be a trifling consideration, but there is much diversity of opinion at the present time regarding the function of tonsils. The best advice which I can garner from the literature is that if the tonsils perform any function after birth it has been accomplished before the age of four years, and thereafter the tonsils cease to be a factor in the human economy and become a rudimentary structure. Hypertrophy of the tonsils, during the period up to four years of age, justifies their removal only when they produce a decided mechanical obstruction. At four or five years of age hypertrophied and cryptic tonsils may justify their removal on the grounds of preventive or expectant surgery. Later in life, the more advanced the age the less I advise removal of hypertrophied tonsils, in the absence of serious infection.

2. Difficulty with nasal breathing.—It must be remembered that inability to breathe through the nose, due to obstruction

caused by tonsils and adenoids, is rarely encountered in older children at the present time. These cases are not allowed to continue as such, in the light of present information and efficient school examination, welfare work and community measures for their prevention. Adults referred for tonsillectomy because of "difficulty with nasal breathing" may be more in need of nasal surgery than tonsillectomy.

3. Snuffles in childhood and evidence of sinus infection.
4. General health.
5. Frequency of colds and bronchitis.
6. Ear complications.
7. Exanthemata and complications therefrom.
8. Attacks of tonsillitis and their sequelæ.
9. Evidence of cervical adenitis.
10. Appetite and disposition.
11. Possibility of hemophiliac.
12. Time that has elapsed since any acute infection and cultures of inflammatory exudate.

Careful consideration of the above points gives one a pretty definite record of any case, and upon the history and information thus gained a pretty definite impression is obtained regarding the pathology to be found.

I cannot emphasize too strongly the cooperation of the internist. In all of my cases a complete physical examination is made just a few days before the operation is performed. To be sure, we operate on cases which show heart and kidney lesions, as in many cases the tonsils seem to be the causative lesion, but not infrequently some acute condition is found of a bronchial nature which renders it advisable to defer the risk of anesthesia for a few days.

In examinations of nose and throat cases attention is first directed to the cervical glands. The presence of enlargements, acute or chronic, with a definite knowledge of anatomic relations, together with the lymphatic drainage, will help to localize the pathology if present. Attention is next directed to the general condition of the mouth, gums, teeth and tongue. A furred, tooth marked tongue is usually an indication of the presence of mouth infection. Should there be present marked irregularities of the edges of the front teeth, a Wassermann examination should be made. The external exam-

ination of glands of the neck and mouth not only gives valuable information, but also tends to inspire the patient's confidence in the examination and dispels much of the fear with which the heart of a youngster, particularly, is filled when anticipating such examinations.

The condition of the pillars, uvula, soft palate and posterior pharyngeal wall is next noted. The information gained from this portion of the examination is of inestimable value. In most cases where infection is present there will be a thickening of the pillars with redness and chronic inflammation of the mucosa covering them. The uvula is often markedly increased in diameter and length, with thickening and redness of the entire soft palate. The lymphoid tissue in the posterior pharyngeal wall is swollen and red, and islets of such tissue are clearly defined. Examination of the tonsil itself consists in noting the size, consistency and cryptic contents. Simple hypertrophy causing mechanical obstruction has already been alluded to. Previous inflammatory conditions will be evidenced by the replacement of normal tonsil tissue by cicatricial tissue and contractions. The cryptic contents should be carefully expressed by pressure against the anterior pillar and the contents carefully noted. If there is any question regarding the substance thus expressed it is advisable to have cultures and microscopic examinations made. It must be remembered that the cheesy masses which are found in 95 per cent of the cases are not pus and must be differentiated from it. If pus is present it is most frequently noted coming from the upper crypts of the tonsil. This the most submerged portion and where the crypts are more nearly closed as a result of their being covered by surrounding structures. One examination is not always sufficient to demonstrate the presence of pus.

Infection in adenoids is not frequently mentioned in literature, but is present more often than generally believed, and is manifested by marked redness and inflammation of the adenoid tissue and structures in close proximity thereto. Profuse nasal secretion often results.

Presence of adenoids results in a variety of symptoms, which must be considered in the same manner as conditions resulting from tonsils. Adenoids must be considered from the standpoint of the obstruction caused by them, the infection

present and their relation to pathology found in surrounding structures. It must be remembered, however, that adenoids are found normally in every individual at birth and have a tendency to atrophy after the fourth year, therefore, their presence previous to the fourth year is not necessarily an indication for their removal unless some good definite reason can be found for so doing.

The benefits derived from the removal of tonsils and adenoids, whether on account of obstruction or a focus of infection, are many, and its effect on clearing up acute and chronic catarrhal infections of the eustachian tube and middle ear is well understood. Where there is found sufficient reason for the removal of tonsils and adenoids, but at the same time there is found hypertrophy of the turbinates or a slight sinus infection, I have made it a rule to remove the tonsils and adenoids and follow these cases from four to six weeks with cleansing treatments and attention to drainage of sinuses and have been gratified to know how many of these cases clear up without surgical nasal interference.

The technic of operation for the removal of tonsils and adenoids must be decided by the personal equation. The operator must find and utilize the method whereby he can remove the structure completely without mutilation of the surrounding tissue and which gives the most perfect postoperative result. To discuss the different methods would be a long story. I have used nearly every method and have finally decided on the scissor dissection as the method par excellence in my hands. By this method the cleanest and most definite separation of the tonsil from the surrounding structures is made and the postoperative results are most satisfactory.

CONCLUSION.

The histologic and bacteriologic examination of 2,321 consecutive specimens of tonsils and adenoids removed during the past four years has developed certain facts and impressions as follows:

1. Tuberculosis of tonsils was found in 39 cases, or 1.7 per cent of all cases examined. This is somewhat lower than the average per cent of other available studies.
2. Syphilis is comparatively rare.

3. Malignancy was found in only one case.

4. The great percentage of tonsils demanding removal for clinical reasons, either because of infection or on account of obstruction caused by their hypertrophy, show either simple hyperplasia or infection with pyogenic organisms.

5. Tuberculosis, syphilis, malignancy, pyogenic and other infections found in tonsils removed impresses the writer with the importance of

(a) Thorough clinical study of the individual case before operation.

(b) Thorough operation to accomplish complete removal, with due respect for the surrounding structures.

(c) Careful study of tissue removed.

(d) Thorough follow up work.

I gratefully acknowledge my indebtedness to Dr. R. R. Mellon for his cooperation in the pathologic work.

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LXIII.

RADICAL MASTOIDECTOMY; COMPLETE HEALING. METHOD OF CONTROLLING PERSISTENT VERTIGO.

BY J. L. MAYBAUM, M. D.,

NEW YORK.

Hugh B., aged 26 years, was admitted to Manhattan Eye, Ear and Throat Hospital, service of Dr. Berens, June 10, 1919, with a history of distressing vertigo and falling, of six months' duration, discharging ears and impaired hearing.

The patient had the usual diseases of childhood. Double middle ear suppuration dated back thirteen years following diphtheria. From then until the onset of vertigo six months ago, except for aural discharge and impaired hearing, there was no complaint referable to the ears. The dizzy attacks at the outset occurred at infrequent intervals until six months before entering the hospital. The attacks then became of greater severity, occurring daily and being accompanied by falling, particularly to the right.

Aural examination disclosed a moderate foul smelling discharge from each middle ear, with perforation and granulations in posterior superior quadrant. Hearing in the right ear markedly diminished, whisper not heard; C (128) and C₄ tuning forks not heard. C—A., 4 sec.; B., 15 sec. Weber to the right. Mixed form of deafness—advanced middle ear deafness with degenerative cochlear changes.

Hearing in the left ear fairly good; whisper, 8 feet; Rinne, negative; high tones as well as the low and middle tones slightly reduced.

There was a spontaneous nystagmus to the left; no spontaneous past pointing. Fistula test negative.

Turning to right—horizontal nystagmus to the left, 20 sec. Turning to left—nystagmus to right, 22 sec.

Caloric stimulation of the right and left horizontal and vertical canals gave normal responses for nystagmus, past pointing and vertigo. Fistula test negative.

I did a radical mastoid operation on the right side. Found a sclerosed mastoid and a cholesteatomatous mass in antrum and aditus and granulation in middle ear removed. The patient left the hospital on the tenth day decidedly better, but still complained of occasional milder attacks of vertigo.

The spontaneous nystagmus disappeared at the end of two weeks, the hearing in the right ear remained as before the operation. The patient came to the clinic for two or three dressings; did not appear again for six weeks. He then returned with the story of having been unable to attend the clinic because of severe attacks of acute articular rheumatism. During this period the ear had been dressed in a more or less perfunctory manner by his family physician. The vertiginous symptoms had recurred with almost as marked an intensity as previous to the operation.

Examination of the operated ear now disclosed an almost complete atresia of the meatus and the canal filled with partly organized granulation and connective tissue. There was a fistula through which a thin purulent foul smelling discharge was escaping.

Hearing very much impaired; could hear only loudly spoken voice with noise apparatus in left ear. Tuning forks not heard. Galton whistle heard.

Caloric test (right ear) ineffective because of the partial atresia of the canal. Caloric (left ear) normal response. A spontaneous nystagmus to the left present. Turning to the right ten times in twenty seconds resulted in horizontal nystagmus to left of 18 seconds' duration. Turning to the left nystagmus to the right 20 seconds' duration.

Patient was readmitted to the hospital. I reopened the post-auricular wound. Cured granulation in meatomastoid cavity and did a plastic operation.

Patient left the hospital in a few days; meatomastoid cavity completely epidermatized in eight weeks. Until the end of May, 1922, the patient continued to complain of vertigo; at times this became so distressing that he had to discontinue his usual activities. Enforced rest gave only temporary relief. The symptoms described by the patient were definitely those of a systematized vertigo. Occasionally these attacks were

accompanied by nausea, but never vomiting. The spontaneous nystagmus ceased three weeks after the second operation and was not again observed.

Functional examination October 11, 1922:

Whisper

Right

0

Left

15 feet

lateralizes to the right

Right

Left

C-A:8 sec. B:25 sec.

C-A:15 B:25

Other forks not heard.

C-6 sec.

Voice heard with noise apparatus in left ear.

Caloric not performed (dry perforation).

Caloric positive in 35 sec.

Turning to right, horizontal nystagmus to left 22 seconds.

Turning to left, horizontal nystagmus to right 18 seconds.

Pastpointing and falling normal.

Normal reactions on testing vertical canals.

Every possible factor causing these attacks of vertigo were considered in order to give relief to the patient.

1. A continuance of irritation from the paralabyrinthine inflammation in the operated ear was constantly kept in mind. With a complete healing of the radical mastoid cavity, the disappearance of spontaneous nystagmus and normal caloric response, this factor had to be eliminated.

2. A paralabyrinthine inflammation in the nonoperated (left) ear was for a time considered. Because of the marked diminution of hearing in the operated (right) ear, I was loath to seriously consider this factor from the standpoint of operative interference. The presence of comparatively good hearing in this ear (whisper 8 feet), together with absence of spontaneous nystagmus, pointed against the left ear as a causative factor. Under treatment this ear has further improved; there had been little, at times no discharge during the past year.

3. Both vestibular nerves and their intracranial pathways gave normal responses for nystagmus, past pointing and vertigo.

4. Cerebrospinal lues has been ruled out, Wasserman being negative.

5. Other extraneous factors, as local infection, gastrointestinal disease, errors of refraction, were carefully considered and eliminated as causes.

6. Vasomotor disturbance of the labyrinth. After unsuccessfully running through the gamut of the various remedies advised for the relief of these patients, such as small doses of quinin, large doses of bromides, pilocarpin, etc., in an empirical way, I began the use of solution adrenalin 1-1000, ten drops T. I. D., by mouth, as suggested by Vernet in his article, "The Treatment of Vertigo by Means of Adrenalin," appearing in the April, 1922, issue of the Laryngoscope. In a few days the patient obtained complete relief from his annoying symptoms. Withdrawal of the adrenalin for a few days after its administration for ten days was followed by a return of vertigo, but to a lesser degree. Adrenalin medication has been discontinued with no recurrence of vertigo. His general condition has steadily improved during the past few months.

1 WEST 70TH STREET.

LXIV.

SOME OF THE ASPECTS OF APHONIA, PSYCHIC AND OTHERWISE.

BY JAMES A. BABBETT, M. D.,

PHILADELPHIA.

The subject of aphonia presents a rare composite of analytic research and routine laryngology. The problem of voice recovery may require the most painstaking study of motor, sensory and ganglionic innervation, investigation of the whole field of metabolism complex, with laboratory findings, and yet equally emphasize the simple office corollaries involved in obtaining the patient's cooperation, suggesting confidence, recognizing idiosyncrasy and, not least, holding the proper perspective toward the factors governing his social and economic efficiency. The field of the topic is purposely a broad one. It is the intention here, however, to study the sequence in aphonia, trace the etiologic factors, analyze briefly the physiologic and anatomic alterations involved and stimulate discussion on the prognosis and remedial measures, not in the fixed aphonias of malignancy, advanced luetic and tubercular dyscrasias, but rather in the psychic and functional types, often exasperatingly fixed in character, and in those arrested structural losses where vocal reeducation, automatic or directed, becomes a possibility—perhaps more concisely expressed, on the basis of etiology and sequence, estimate the balance of possibility in restoration.

From the anatomic and physiologic standpoint the mechanism of phonation depends upon the sliding and rotation of the three surfaced arytenoid cartilage to whose anterior or vocal process the cords are attached, and to whose external or muscular process the intrinsic musculature is inserted. To briefly review, the cricothyroid with more or less vertical and horizontal fibers tightens the cords by the raising of its anterior and tilting downward of its posterior half. This muscle practically alone receives its innervation from the external branch of the superior laryngeal nerve. The internal branch

of the superior laryngeal affords sensory supply to internal surfaces of the larynx, including the ventricular space of Morgagni—true and false cords. Its antagonist is the thyroarytenoid muscle which has two planes, internal and external, which may act independently in the falsetto voice, which might perhaps be considered a threshold to aphonia. The cricoarytenoid posticus and lateralis, by external and internal rotation of the arytenoid respectively abduct (widen) and adduct (narrow) the rimal slit. The posterior arytenoid crossed and horizontal fibers draw the arytenoids together and complete the tightened closure. All of these latter muscles depend upon the recurrent laryngeal for innervation.

Holmes Jackson's careful analysis of the "Reflexes From the Larynx" shows the complexity of neurologic factors involved and emphasizes the corollation of the glossopharyngeal and respiratory center in the medulla. Careful study of this mechanism makes it clear that in the types under discussion complete aphonia can scarcely be separated from partial and intermittent phases, for we are considering types capable of voice restoration. This may include deviation in muscular structure, vocal cord surface and any encroachment upon intralaryngeal space as well as nerve integrity.

Etiologic factors in aphonia, while in general of central, peripheral and toxic origin, as grouped by Lederman, present a marvelous complexity of basis factors. In the central group might be included bulbar lesions, thrombosis, embolism and hemorrhage, brain tumors and intracranial growths at base, multiple and lateral sclerosis, parasyphilis and syphilitic gummata, glossolabiolaryngeal paralysis, tabes dorsalis, lesions involving the vagus and recurrent, every form of cervical pressure and trauma, mediastinal irregularities of varied sort, aneurysms, pericordial and peribronchial effusion, enlarged thymus, diseased thyroid, constriction of postoperative scars and ligatures, pleural thickening in apex, principally right, dilatation of left auricle, diseases of mediastinal glands, carcinoma, sarcoma, lymphosarcoma, syphilis and tuberculosis, involvements of esophagus, cervical adenitis, etc. Dr. Cress has reported from recent Vienna clinic observations additions to this list.

From a practical standpoint, the third and toxic group have a more practical application to the laryngologist in our present emphasis upon focal infection—varied toxins of mineral and vegetable origin, lead, arsenic, potassium iodid, iodoform, alcohol, morphin, crude opium, cocain, belladonna; toxins from the acute infectious diseases—pneumonia, typhoid, diphtheria, puerperal fever, measles, gonorrhea, influenza, acute rheumatic fever, disease of thyroid, metastasis from infection of tonsils, teeth, sinuses, middle ear, broken down cervical glands, with more often perhaps streptococcic infection. Under this heading might be included poisoning from inhaling gases, of which so much postwar history has been quoted by Milligan, Hurst and others.

Tuberculosis must be given an important place, from both its mediastinal influence and its contribution to the asthenia so common in hysterical or functional aphonia. Garland and White reported, in 1920, a series of nine cases of paralysis of the left recurrent laryngeal associated with mitral stenosis, in the Massachusetts General Hospital during the past eight years. They consider the record of only 61 cases in the literature, including theirs, as indicative that many are overlooked. A great variety of special etiologic types have been also recently reported, such as: Eversion of the Ventricle and Saccule, by Moore and Tapia; Relaxation of the Diaphragm, by Stoerck; Amyloid Degeneration, by Bruzzi, Evers, Ackerman and Harbitz. Time prevents further enumeration.

With this basis of fundamental mechanism and complex group of etiologic factors, it is our purpose to discuss three important aspects in aphonia, quoting one or two selected illustrative subjects: (1) The metabolism complex, or syndrome in aphonia; (2) the borderline difficulty in apparent psychic types; and (3) the phonation prognosis after arrested tissue loss.

1. The Metabolism Syndrome.—Gill in a recent address before the section in Philadelphia emphasized the importance of the metabolism factors in arthritic cases which per se should have been relieved by surgery of the teeth, infected tonsils and sinuses—with such a manifold group of etiologic factors as we find active in types of aphonia often not simply central, peripheral or toxic, but varied combination of these, pro-

foundly subject to endocrin and basal metabolism influence, it is reasonable to link functional aphonia to general metabolism. From the simple aphonia of acute laryngitis to the paralysis from recurrent laryngeal bloc, at least circulatory metabolism is impressive. The nature of a nerve impulse is obscure, the chemistry of its root cell not much less so, but trophic influence of nutrition upon both will be admitted. Toxemia, zymolysis, acidosis, endocrin changes must affect this neuromuscular action, and particularly in myopathic types. The carbohydrate balance regulation of the posterior pituitary lobe grow stimulation and sexual activity, control of the anterior, have a reasonable relation to psychic aphonia.

One of the reasons for the inception of this paper was the difficulty experienced in recent years in determining the basis of aphonia in borderline cases, and to stimulate discussion upon the same the following case is cited for illustration:

Case 1.—In July, 1920, Carrie G., referred by Dr. B. Girl of 15, slender, phlegmatic rather than neurotic in tendency. Early history negative except for series of spasms reported at three years of age and suppurative otitis media, right side. Somewhat later, under medical care for reputed cardiac weakness. Voice gradually disappeared nine days prior to visit. Upon examination, larynx apparently normal. August, 1920, tonsils and adenoids removed at Lankenau Hospital. September, 1920, digital avulsion of cords, resulting in modified phonation half of one day. Physical, neurologic, fluoroscopic, laboratory urine and blood examinations all negative. Tonics, thyroid, pituitrin and ovarian extracts and iron injections administered. No result. Wassermann negative. Massage and electricity ineffectual. In December, 1922, a typical reflex cough appeared, apparently due to recurrent laryngeal irritation, and persisted for three or four weeks. During this time the laryngoscopic picture presented a normal appearance. Adduction and tension bilaterally equally failed to produce tone. Cords were apparently approximated, though somewhat weakly, and lower triangular area sometimes remained open. According to Semon-Rosenback law, indications all point to functional aphonia.

The significant premise in this recital is that the voice has failed to return except for this one-half day, and now and

then a very incidental approximate tone, for a period now of almost three years. Within the past month a further somewhat exhaustive study has been made. Larynx still appears somewhat weak. No obvious fault in the laryngeal cavity. Careful X-ray examination by Dr. Pfahler shows sinuses frontal, sphenoid, maxillary clear, slight amount of exudate in ethmoids. A somewhat deflected septum and spur may be related to this, but patient declines operative relief for this. Sella turcica normal. No constriction of the trachea from pharynx downward. No mediastinal tumor or gland. A few calcified lymph nodes in mediastinum about hilus of lungs but no evidence of pulmonary disease. Heart and aorta normal. External examination of chest and heart also normal. Barany tests were taken in intercranial search. Horizontal rotation nystagmus, past pointing and vertigo normal. Douching right ear with cold water—vertical position, no response for five minutes; with head back, slight horizontal nystagmus appeared. Douching left—good response, 1 min. 45 sec. Ocular, falling, Rhomberg and reflexes normal. Recent blood examination and basal metabolism all negative; erythrocyte count, however, 4,000,000.

These reports have been only briefly reviewed to cite a typical case of difficulty in borderline cases. With one exception (of Barany rotary nystagmus after douching in an ear where acoustic labyrinth was unaffected) all symptoms point to functional aphonia, yet one in which the metabolism complex apparently aids in rendering the aphonia static. The serious influence in the patient's life regime is obvious. The matter of its organization would be simple were the basic evidence of some central difficulty or pressure neurosis. The case seems a complex involving perhaps endocrins and peripheral circulation and idiopathic asthenia, and would perhaps be grouped as a case of type reported by Stern, photoasthenia.

The concluding aspect of aphonia to be considered is that of the natural effort of the larynx and its associated structures to get well. The marvelous results in reeducation of the voice after total laryngectomy impress the possibility in this direction. The Early case reported by Cohen and the experiences of varied operators more recently attest this farther. In illustration of this phase the following history may serve:

Case 2.—In March, 1922, was called to see patient, Mr. J., who had developed increasing difficulty in phonation, and even some dyspnea without pulmonary complication following a major abdominal operation. The abdominal operation was reported as malignant, requiring removal of a segment of intestine—in fact, a portion of the malignant area was inoperable. The patient had some indefinite previous laryngeal history. As his legal profession demanded much public speaking, his previous trouble was probably of no organic nature. Certainly there was no specific, malignant or tubercular history locally.

In recovering from the above cited condition, at which time the stomach tube had been used, the patient experienced excessive retching and vomiting, and to control this repeatedly clutched the throat and larynx with finger far back in the pharynx, infectively and traumatically complicating the picture. The patient when I visited him had complete aphonia and rather alarming dyspnea, and his larynx presented a picture of general congestion, edema, mucopurulent secretion and irregularly dislodged slough, entirely filling the ventricular cavities and obscuring all view of cords and arytenoids.

Careful examination by cultures and smears only revealed mixed infection largely staphylococci, and no t. b. bacilli were found. Under local treatment, administered with care on account of edema and dyspnea, the congestion gradually subsided, first right and then left cord came into view and finally the arytenoid area. In the meantime Wassermann test was taken and found negative. Voice gradually was recovered, though left cord showed apparent tendency to abductor paralysis. In breathing, the position was usually miocadaveric, though the article by Fein would deny this term.

On March 28th, eighteen days after the original examination, patient coughed out what appeared to be left arytenoid cartilage, and, two or three days later, the right. X-ray, fluoroscopic and laboratory examination confirmed this diagnosis. Patient was profoundly aphonic at this time, though dyspnea had improved. About three weeks later patient commenced to recover voice, and in six weeks produced fair tone, low pitch for the most part, and variable in force. In June the patient was placed under care of a New York laryngologist,

his larynx capable of moderate tone production, alteration in the arytenoid field, but no malignant evidence. On November 9th, reported somewhat discouraging reversion to whispered or modified phonation, but patient decidedly asthenic from intraabdominal progress in malignancy.

This case is of interest from the fact that he was recovering acceptable voice in spite of the absence of both arytenoids. The literature is replete with instances of recovery of voice after removal of one or even both cords and total laryngectomy, with subsequent voice reeducation. Prof. Chiari reported in 1909 before the society his results after operations for malignance. Time will not permit us to discuss this in further detail.

CONCLUSIONS.

1. Partial and intermittent types of loss of phonation may properly be considered with aphonia and the latter not considered a fixed aphonia except in a limited group of conditions.

2. The complex changes of metabolism, variation in endocrins, zymolysis, acidosis, toxemia, etc., may profoundly influence functional aphonia.

3. The complex symptomatology of borderline cases, organic vs. functional aphonia, renders diagnosis and prognosis extremely difficult.

4. The reconstructive forces in restoration of phonation after arrested pathology, even with marked tissue loss, are most surprising.

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LXV.

FOREIGN BODIES IN UPPER AIR AND FOOD
PASSAGES—A CLINICAL REPORT.*

BY HARRINGTON B. GRAHAM, M. D.,

SAN FRANCISCO.

What Jackson said in 1915 concerning the armamentarium in bronchoscopic or esophagoscopic work is still pertinent in spite of the advances made in the mechanics in this field. He then said that there was no one set of instruments which could be considered the best for all work or for all men; that it was more the man behind the instruments that was going to save life, rather than the instrument itself. If one has at his disposal the large tubes of Mosher as well as the small ones of Jackson and has practiced with both, he will certainly choose the Mosher tubes for the examination of an esophageal carcinoma in an adult and the Jackson tubes for a foreign body in the bronchus of an infant.

The advantages of a proximally lighted tube for diagnostic purposes are, it seems to me, beyond a doubt but the disadvantage in the dispersion of light from an instrument introduced into this tube during the removal of a foreign body tends to render it far less serviceable than the distally lighted tube. Skill in the handling of any of the instruments can only come with painstaking practice, and the best results will certainly follow through a skill acquired in the use of a variety of instruments. Our ultimate purpose is to save life with the least damage to the tissues involved and with the least strain on the patient; not to satisfy our egotism by a claim that a foreign body has been removed with the smallest tube in the shortest time on record. If by using a small tube the patient is subjected to three seances, whereas had a larger tube been used, it could have been removed in one, it were better to have chosen the larger diameter tube. Each case must be judged

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by itself, and that method or instrument selected which will best fit the circumstances and conform to the skill of the operator.

In analyzing my cases, I have been surprised at the large number of unorthodox methods used, and still out of 43 cases of foreign bodies there has been but one death, a mortality of 2.3 per cent or 5.8 per cent of the bronchoscopies. For instance, in two cases (Nos. 1 and 32) I resorted to a low tracheotomy in order to reach the foreign body; in both cases a prolonged effort had been made to get the article and in each case it was readily extracted through a tracheotomy opening. Again a fluoroscopic bronchoscopy was done twice, once unsuccessfully and once with a most satisfactory result. The successful case (No. 33) had been tried under local anesthesia a year before, but there was such a large quantity of pus present that there was but little chance of locating the article, a shirt stud, by vision. Under ether with the fluoroscope a tube was passed readily to the article and later a forceps which was closed upon the stud and a successful result obtained. The stud had been in the left lower lobe for twenty-three years and granulation tissue had formed over it. No effort was made to remove the granulations, as Jackson had done in a similar case, as the probe passed so easily. At no time was the foreign body seen through the bronchoscope, but the forceps seemed to enter a cavity when passed to it, and there seemed to be little chance of grasping lung tissue.

Unorthodox methods do not necessarily mean poor judgment. They may mean a less skillful technic, but perhaps a more rational safety for the patient. I have seen a little patient go back to bed in thorough shock after a struggle with the overzealous staff of physicians who were attempting to pass a tube without an anesthetic, whereas a little ether would have quieted the nerves of both the physicians and bystanders, to say nothing of the patient. It is one of the triumphs of surgery that this can be done so easily by the skilled endoscopist. It is one of the tragedies that it should be tried by the less skilled in a well intentioned but ill advised effort to save the child or to show his prowess.

There have been foreign bodies which, if dislodged, would close off the only lung tissue which was functioning where it

was extremely desirable to operate without an anesthetic, but these cases are few and far between, and I believe with the bronchoscope in such universal use as at present that it would be far wiser to advise the use of an anesthetic until the requisite skill had been acquired. After the tube has once been introduced we all have found that it takes but little ether to continue the work indefinitely, especially if cocain is used at the distal end of the tube. All of my cases under 6 years of age, save one, two cases of the five between 6 and 20 years of age and two of the 25 above 20 years of age were etherized. All of the others were cocainized save a baby of 8 months, on whom an esophagoscope was passed. For diagnostic or treatment purposes strictures, carcinoma, thyroid, asthma, a number of cases have been examined without an anesthetic but with indifferent success.

In the diagnoses of these cases some interesting facts were brought out. In five cases (2, 21, 27, 32, 38; metal whistle, gold crown, primer, pine nut, blanched almond) a whistling could be heard, in three near the patient and in two across the room; in each case it had been observed by the family before seeking the doctor. This whistle immediately disappeared on removal of the foreign body. It has been the solid bodies which nearly occluded the main bronchus which apparently produced this whistling sound.

In Case 3 the patient insisted for years that he had a tack in his lung but was laughed at by his doctors. It remained in twenty years, causing hemoptosis, night sweats, pus expectoration and fever.

Case 33 was accepted in the army after an extensive lung examination, including a radiologic examination on account of the claim of the patient. After entering the army further X-ray pictures were taken with a negative result. After discharge from the army, at which time he says he was declared to have had normal lungs, he developed a lung abscess and fell into my hands. A radiograph showed clearly the metal shirt stud in the left lower lobe bronchus of about the fourth degree.

In Case 32 the radiograph showed an uneven edge at the top of the primer which should have been interpreted as a

closure of the top. The error in not so interpreting it cost the child a number of seances. Too much stress cannot be laid upon a prolonged and careful study of every element connected with the diagnosis of the character, size, shape and location of the intruder before any attempt is made whatever at removal, for at times the simplest element may be the secret that effects the solution of the problem. A hurried diagnosis and an eagerness to get at the case has been the only cause, I believe, of many a failure. The mere presence of a foreign body is seldom a tragedy, but a failure to get it at the first sitting may be.

A diagnostic symptom which has been held of value in these cases is a sudden spasmodic cough in a child which has been previously well, but this sign, pronounced in its suddenness, was present in a child of two years in whom no foreign body was found and who passed through a typical attack of lobar pneumonia, so that one cannot rely entirely on such a history.

Special points of interest noted in the treatment of this series of cases are:

Case 17.—A gold clasp pin lying open in the left bronchus of a nine-months-old child was so manipulated as to bring the point of the pin into the tube; the body of the pin was then grasped and pulled through the tube. For two days there was a temperature of 101 to 103 degrees, and considerable difficulty in breathing, relieving by a steam tent. Subsequently the child made an uneventful recovery.

Case 18.—A case of an open safety pin in the upper third of the esophagus of an eight-months-old child. Again the point of the pin was brought into the lumen of the tube, the body of the pin grasped and the pin partially closed and the tube and pin brought out together.

Case 23.—The open safety pin was at the lower end of the esophagus in an eight-months-old child. I had no pin closer which would pass through the tube, and as the surroundings were unfavorable I judged there would be more trauma in an attempt to extract perorally than there would by an incision through the abdominal wall. I therefore passed the esophagoscope after an incision had been made and pushed the pin down onto the finger of the surgeon. The whole operation took twenty minutes and the child made an uneventful recovery.

Case 22 was that of a bullet in the antrum. The surgeon, while operating under ether with a fluoroscope, lost the lead, which dropped into the right bronchus. I was called to the hospital and introduced the tube while the patient was still under the ether. It was a strange and fortunate coincidence that where a fluoroscope was so necessary it was being used for another purpose.

Case 24 was a series of tragedies. The patient, an adult, had swallowed a chicken bone, which had lodged in the upper portion of the esophagus, and after three days produced an abscess in the neck. The bone was removed under local anesthesia, but in so doing the porcelain tip of an incisor tooth was broken off and slipped outside of the tube into the abscess cavity. Three unsuccessful efforts were made to reach it perorally, during one of which the tip of one of the forceps broke off, lodging in the esophagus and necessitating its removal. An effort was now made to reach the porcelain through an incision in the neck. At no time were we able to feel the article, but the manipulation evidently forced it into the esophagus, as an X-ray subsequent to our operation showed an absence of the porcelain. An ill wind seemed to be following this patient, as, while I was working on her, her mother aspirated a small fruit pit and was about to submit to a bronchoscopy when, after three days, she coughed it up.

Case 25 was a wishbone which had been pushed into the larynx by digital manipulation, lodging in the sinuses of Morgagni in such a way as to be nearly invisible to the mirror. It was extracted by the indirect method, causing a sensation of astonishment to the operator as it gradually unfolded itself. Part of the bone was broken off after extraction, so it does not show in toto in the illustration.

Case 27.—A gold crown was found in the left bronchus of an adult with the open portion up so that it was easily grasped. It would not pass through the tube, however, and caught at the glottis. An effort was made to manipulate it through the glottis, during which it slipped off the forceps, dropping fortunately into the esophagus, and was promptly swallowed.

Case 32.—A child of 4 years, with a 22 primer in the right bronchus, had four bronchoscopies (three by myself) without

the operators seeing the foreign body. The only explanation I can give is that the primer was lying close to the lower wall and fitted so snugly that it appeared to be the carena itself. Through a low tracheotomy and a large tube there was no difficulty about locating and removing it.

Case 39 had been shot in the face fifteen years before and had not known that he carried the lead about with him. He came to me complaining of a soreness in the posterior pharyngeal wall, and an X-ray revealed three pieces of lead, one of which was producing an ulceration. This one was easily removed but the other two were not located even under ether.

Case 40, while driving a machine on the highway, suddenly was cognizant of a stinging sensation in the throat and an inability to talk. On examination it was found there was blood on the skin over the larynx, and he was taken to the hospital. An X-ray revealed a 22 bullet imbedded in the larynx wall, evidently a chance spent shot. A surgeon made an unsuccessful attempt to remove it through an external incision when I was permitted to see the case. To date the body remains in situ. An unsuccessful bronchoscopy was done by another surgeon in an effort to get the article.

Case 41.—A child of 15 months had inspired a tack, which had lodged in the right bronchus, and the X-ray showed it at the level of the diaphragm posteriorly. A bronchoscopy did not reveal the tack, and the child developed a pneumonia, from which she died five days later.

Case 42 came to the hospital eight days after having swallowed a furniture tack. One attempt had been made to get it, but unsuccessfully. X-ray revealed the tack in the left lower lobe bronchus. There was a temperature of 104, with remission to nearly normal each day, and signs of pneumonia, with leucocytes of 26,000, so that there was a delay of five days before a bronchoscopy was attempted. As the temperature did not go down a successful attempt was made to extract the tack, the temperature immediately dropping to normal and remaining there.

The first fourteen of these cases were reported in the California State Journal of Medicine, September, 1916.

	Age	Foreign Body	Time of Sojourn	Location	Tube	Anesthetic	Forceps	Result	Time	Route	Remarks
15. Dr. E.	30 yrs.	Fish bone	2 hrs.	upper $\frac{1}{3}$ esophagus	10	cocain	plain	removal	5 min.	peroral	
16. Griffin	30 yrs.	chicken bone	2 hrs.	upper $\frac{1}{3}$ esophagus	10	cocain	plain	removal	5 min.	peroral	
17. Chollote	9 mo.	gold beauty pin	4 da.	l. lower lobe bronchus	7	chloroform	plain	removal	20 min.	peroral	Difficulty in breathing following removal.
18. Zeller	8 mo.	open safety pin	24 hrs.	upper $\frac{1}{3}$ esophagus	8.5	ether	plain	removal	20 min.	peroral	Tube passed by pin once.
19. C. D.	12 yrs.	rubber pencil tip	7 da.	l. l. lobe bronchus	10	ether	plain	removal	10 min.	peroral	Attempt made by another surgeon.
20. S. C.	8 mo.	open safety pin	2 da.	esophagus	7	none		not seen passed into stomach			Baby passed pin in stool.
21. Kalsbeek	1 yr.	blanched almond	14 hrs.	trachea at bifurcation	7	ether	plain	removal	8 min.	peroral	Attempt made by another surgeon.
22. D. C.	30 yrs.	lead bullet	1 hr.	right lower lobe bronchus	10	ether	plain	removal	10 min.	peroral	Accident occurred while under ether.
23. E. C.	3 yrs.	open safety pin	32 hrs.	lower $\frac{1}{3}$ esophagus	8.5	ether	plain	removal	20 min.	gastro-enterostomy	Pin pushed into stomach.
24. Miss B.	30 yrs.	forceps chicken bone tooth	5 min. 5 da. ?	upper $\frac{1}{3}$ esophagus	10	cocain	plain	removal			Three foreign bodies in one case.
25. Eyre	30 yrs.	chicken bone	22 hrs.	larynx		cocain		removal	10 min.	peroral	Removed by indirect method.
26. Mrs. F. Smith	25 yrs.	chicken bone	23 hrs.	esophagus	10	cocain	plain	removal	5 min.	peroral	Removed by the cocaineizing brush.
27. Meloney	40 yrs.	gold crown	2 hrs.	l. l. lobe bronchus	10	cocain	plain	removal	10 min.	peroral	Crown dropped into esophagus.
28. Edwards	12 yrs.	water-melon seed	3 da.	l. lower bronchus	8.5	cocain	plain	removal	10 min.	peroral	

	Age	Foreign Body	Time of Sojourn	Location	Tube	Anesthetic	Forceps	Result	Time	Route	Remarks
29. V. C. 1	25 yrs.	pin	1 da.	l. l. lobe bronchus	10	cocain	plain	removal	5 min.	peroral	
30. V. C. 2	20 yrs.	chicken bone	2 hrs.	middle $\frac{1}{2}$ esophagus	12	cocain	plain	removal	5 min.	peroral	
31. V. C. 3	25 yrs.	meat	1 da.	esophagus	12	cocain	plain	removal	5 min.	peroral	
32. Riley	4 yrs.	primer	4 wks.	R. l. bronchus	7.5	ether	plain	removal	4 attempts 1 hr. each	tracheotomy	Once attempt by another surgeon.
33. Archibald	24 yrs.	shirt stud	23 yrs.	l. l. lobe bronchus	10	ether	double curette	removal	2 attempts		Under fluoroscope, former attempt by self.
34. Graham	3 yrs.	25c	3 hrs.	esophagus	8.5	ether	plain	removal	10 min.	peroral	
35. Lamb	50 yrs.	chicken bone	12 hrs.	esophagus	10	cocain	plain	removal	5 min.	peroral	
36. F. P., 1	20 yrs.	fish plate	2 hrs.	esophagus	12	cocain	plain	removal	5 min.	peroral	
37. F. P., 2	30 yrs.	fish plate	12 hrs.	esophagus	12	cocain	plain	removal	5 min.	peroral	
38. Cecchini	8 yrs.	pine nut	4 da.	right bronchus	8.5	cocain	plain	removal	10 min.	peroral	Whisper heard with mouth open.
39. C. K.	38 yrs.	lead bullet 3 pieces	15 yrs.	post pharyngeal wall		ether cocain		2 removals		peroral	The third could not be found.
40. Engelsted	30 yrs.	spent bullet	2 da.	larynx wall		ether		not found			Patient seen in consultation. No attempt by self.
41. J. Miller	15 mo.	lark	5 da.	r. l. lobe bronchus level diaphragm	7	ether		died 5 days pneumonia	1 hr.		Tack not seen.
42. Rule	7 yrs.	lark	13 da.	r. l. lobe bronchus	8.5	ether	plain	removal	15 min.	peroral	Fever of 104 with pneumonia. Fever dropped with removal.
43. Murphy	40 yrs.	tin bottle cap		upper esophagus		cocain	double curette	removal	1 min.		Indirect method.

THE COCHLEA-PALPEBRAL REFLEX IN INFANTS.*

BY THEODOR DEMETRIADES, M. D.,

ASSISTANT ON THE EAR, NOSE AND THROAT DEPARTMENT
OF THE UNIVERSITY POLICLINIC,

ATHENS, GREECE.

The question as to whether or not the newborn hear is dependent upon observing the reaction which follows a sound irritation on the ear. By different investigators this question has, however, been answered in many different ways, due to the fact that different methods of investigation were used in reaching these conclusions

Among the first to investigate this subject must be mentioned Kussmaul, who in a short contribution held "that of all the senses, hearing was the longest to remain dormant." Genzer and Lange held that during the first and second year of life the reaction to acute irritation was already present—in fact, the movements of the whole body but not the cochlea-palpebral reflex. In like manner as both of these authors, Moldenhauer held to the same views. On the other hand, Poli holds that a ten-day old child will respond to a noise; however, he does not specify the distance for this form of reaction. Sachs gives the appearance of the hearing in a child at 7, Gomperz at 1 year old. Kohlreuter could obtain no reaction in the first 24 hours after birth by the use of tuning forks (C_1 , C_2 , C_3), but could obtain a definite reaction from the Galton whistle. In like manner, Zimmermann arrived at the same conclusions.

Prever holds to the opinion that all newborn are deaf, with-out exception, both to weak and high tones as well as to noises,

Translated by Dunbar Roy, A. B., M. D., Atlanta.

*From the Ear Department of the Policlinic in Vienna. Service of Prof. G. Alexander.

The translation of this article was undertaken because the subject is not mentioned in our textbooks on Otology and Ophthalmology. It has seemed of so much importance to me that I feel as if the article should be placed in readable form for English-speaking Otologists. (D. R.)

and that the first reaction to sound irritation appears on the fourth day.

Kutvirt has been one of the most thorough investigators of the hearing changes in the newborn. He proved that while testing the hearing of the newborn (using C_2 and C_3 tuning forks) he was able both to arouse the infants from sleep or to produce crying or to call forth some general muscular reaction. The aural palpebral reflex was only incidentally investigated by this author, without attaching any importance to the same.

On the other hand, an objection must be raised to these tests by Kutvirt, because of the fact that on account of the various degrees of sleep from which these infants were aroused, the hearing irritation would not work the same in all cases. From these experiments Kutvirt came to the conclusion that the newborn does show reaction to the hearing stimulations at various times after birth and, in fact, he observed them one-half hour after birth of the child.

The varying intervals of nonresponse to hearing stimulation, according to Kutvirt, stands in close relationship to the length of the childbirth, and on the ground of his statistics he holds that the longer the interval between the first pain and the birth of the child, the later will the hearing reflex appear. From a survey of the above literature it is evident that up to this time only the general body reactions had been considered as indicating the presence of hearing in the newborn. Now, however, the recent investigations have hinged on the importance of the aural palpebral reflexes as a diagnostic sign for determining the presence of hearing. The question therefore is the relationship of this reflex to the newborn.

Encouraged by Prof. Alexander and having had access to the rich material of the II University Woman's Clinic through the kindness of Prof. Weibel (to whom I hereby acknowledge my thanks), I have tested out the possibility of hearing in 105 newborn in order to determine how long after birth is the appearance of the cochlea-palpebral reflex. In reference to the experimental tests the following is to be noted:

The investigations by Cemach, Wotzilka and Brunner show that positive results from the cochlea-palpebral reflex are

present only with persons who hear, otherwise it would be impossible to draw fixed conclusions.

From a negative result of the reflex, as Cemach has always held, we should be very cautious in drawing a conclusion because it can be absent even in those with normal hearing (8 per cent of cases).

Proceeding now to my own studies as to the results obtained in these investigations, we come to the following conclusion:

If the cochlea-palpebral reflex is produced in a child then it is evident that the child possesses some hearing, but this means that it indicates nothing as to the future degree of hearing in the examined person.

The cochlea-palpebral reflex was produced by striking the tuning fork C_4 with a small uncovered percussion hammer against the metal prongs. Just opposite to the method pursued by Kutvirt I tried to get the children for examination while they were fully awake. This was accomplished by the nurse holding the child on her arm and playing with it or the child was awakened out of its sleep by my passing a small ear speculum into the external canal. The tuning fork was struck four or five times close to the ear of one side at intervals of about 15 seconds. The appearance and the intensity of the reflex seemed to be dependent upon the age, the weight and the size of the infant.

The type of reaction was not the same by all children. Some reacted with distinct opening and closure of the eyelids while others moved the whole body as if frightened. Most of the children began to cry following the striking of the tuning forks. In none of the 105 newborn who were examined did the reflex appear immediately after the cutting of the cord (10 cases); the reflex appeared most frequently between the second and third hours after birth (Tables I and II), but usually from the fourth to the sixth hour after birth.

This statement is only in reference to children whose birth was normal and whose parents were healthy. At the latest the reflex appeared during the first 48 hours after birth.

If within this time the reflex could not be produced, usually it was not possible to make it appear even within the next eight to ten days (two normally born children, healthy par-

ents). After this time I could not keep the children under observation because they left the clinic with their mothers.

TIME OF THE APPEARANCE OF THE COCHLEA-PALPEBRAL REFLEX
AFTER BIRTH.

TABLE I.

Number of hours after birth in which the reflex appeared for the first time.	Number of children show- ing reaction.
2 Hours	1
3 Hours	2
4 Hours	15
5 Hours	24
6 Hours	26
7 Hours	2
8 Hours	3
9 Hours	2
10 Hours	1
11 Hours	1
13 Hours	1
15 Hours	1
16 Hours	1
19 Hours	1
22 Hours	2

TABLE II.

Number of days after birth in which the reflex first appeared.	Number of children show- ing reaction.
1 Day	3
6 Days	1
7 Days	3
8 Days	3
9 Days	3
11 Days	2
12 Days	1
13 Days	1
16 Days	1
19 Days	1

From the investigations of the previously mentioned authors, as well as from my own results, it must certainly be concluded that the cochlea-palpebral reflex is absent during the first few hours after birth.

For an explanation of these results various authors have attributed different causes.

Most of the investigators attribute as the cause for the absence of hearing, to the filling up of the auditory canal with the vernix caseosa. Gomperz and V. Urbantschitsch think that it is due to the narrowness of the canal in the newborn.

According to Vierordt, the late appearance of the hearing in infants is due to the horizontal position of the drum membrane; according to Kutvirt, to the poor elasticity of the membrana tympani, as well as to the edematous material in the middle ear which is known to exist in infants. It is evident, therefore, that these sound conducting impediments are the cause for the late appearance of the hearing function.

If we accept the correctness of these explanations then we must draw the conclusion that the hearing of the newborn will first be evident when these various impediments in the sound conducting apparatus have been removed.

Such a thing is improbable because my previous investigations have shown that the cochlea-palpebral reflex appears as a rule much earlier than the impediments to the sound conducting apparatus are supposed to have disappeared. Moreover, it has already been shown by Kutvirt and Gomperz that the late appearance of hearing in the newborn could be explained by the incomplete development of the inner ear and brain.

However, this explanation cannot be entirely satisfactory, since we know that the labyrinth as well as the brain of the newborn is morphologically completely developed, if we mean the structure in the neighborhood of the acoustic fibres and center for hearing.

In cases of premature birth the conditions are naturally different and it is possible for the hypothesis of Gomperz and Kutvirt to be accepted in such cases. This is also true in cases of hereditary syphilis in the newborn.

In his second communication on this subject Kutvirt, as before mentioned, found a relationship of the reflex to the lengthened time of labor pain to the expulsion of the after birth.

He holds to the belief that in the newborn whose birth was associated with both protracted labor and late reflexes the head

in being held back was exposed thereby to increased pressure. I believe, however, that this obstetrical condition is not the most important cause in the delayed reflexes, for it must be remembered that, according to the statistics of Kutvirt in those cases which were delivered by Caesarian section after a long but tedious labor, the reflex appeared earlier than in those in whom the birth was only thereby retarded, as for example in breech presentation where the fetus was delivered by turning. From these circumstances it follows that the delay in the appearance of the reflex in the newborn in breech presentation was due to the increased compression on the skull in consequence of the increased labor pain. Moreover, infants who were delivered by Caesarian section had also suffered more from this greater compression. Therefore, we must the more, as I believe, attribute the late appearance of the reflexes in those cases of breech presentation to a longer and more continuous asphyxia of the after birth, and this must also cause some interference with the functions of the brain. We know, moreover, through Verworn, who has investigated the subject, that the brain is exceedingly sensitive to the withdrawal of oxygen.

From the above it is evident that Kutvirt, according to his observation of the newborn who suffer with a complicated labor and when the hearing reflex was late in developing, has only recognized the well known fact that the hearing reflexes can only be tested on the brain, possessing its complete function.

I have therefore undertaken to prove again these observations of Kutvirt.

Finally, it is well to note the abnormal changes following this reflex.

1. In two cases of normally born children of healthy parents the reflex was absent during the whole time while they were under observation.

2. In five cases of new born of syphilitic parents the reflex was likewise absent. It must be noted, however, that in the last mentioned cases the reflex appeared two to three days after the birth. On the other hand, I was able in one case to produce the reflex even 16 hours after birth. An earlier appearance of the reflex I was not able to observe.

3. In one infant where the mother suffered from eclampsia the reflex was able to be elicited for the first time three days after birth. This artificially delivered child had a body weight of 1800 gr.

4. In one child with scrofulosis which lived six days, no reflex could be obtained.

5. In one child born asphyxiated the reflex during the first two days could not be elicited.

6. In those of premature birth, i. e., seven or eight months, the reflex appeared six to eight days after birth, yet I was able in one premature birth, whose height was 43 cm. and weight 1805 gr., to obtain the reflex 15 hours after birth.

What especially concerns those cases of premature births is given in the following results:

Of the 18 premature births which had been studied, the reflex could be elicited in ten cases, between the sixth and ninth day after birth, in one each of the premature births; in the others, first on the 12th, 13th and 19th day after extrauterine life. Two of the premature births left the clinic on the eighth day and up to the time of departure no reflex was observed.

Of the two remaining premature births, the reflex appeared in one of them on the first day and this continued until the infant left the clinic. In the second case the reflex up to the 9th day of extrauterine life could be elicited and then it disappeared.

In all the cases with negative cochlea-palpebral reflex the appearance of the drum as well as the external meatus appeared normal by otoscopic examination. Among the premature births three children were found to show hereditary syphilis.

EXAMINATION HISTORIES OF THE PREMATURE BIRTHS.

No. 3607. Caroline Sp. History: Measles. Abortions 1906, 1908 and 1909. First pains began Dec. 17th, 3 o'clock a. m., birth 3:15 p. m. Length 48 cm., weight 2070 gr. Spontaneous birth. Reflexes appeared 16 hours after delivery.

No. 3681. Wilh. St. History: Rachitis. Pains began Dec. 23rd, 11 o'clock: birth at 4 p. m. Boy. Length 49 cm.,

weight 2520 gr. Spontaneous birth. Appearance of reflexes in 6 days.

No. 3451. Maller, Chr. History: Scarlet fever. Pains began Dec. 4th at 12 o'clock noon, birth 2:45 a. m. Boy. Length 48 cm., weight 2630 gr. Spontaneous birth. Reflexes 7 days after birth.

No. 3459. Leop. D. History: Measles. Pains began Dec. 6th, 2 o'clock a. m.; birth at 3:15 p. m. Girl. Length 48 cm., weight 2700 gr. Spontaneous birth. Reflexes 7 days after birth.

No. 3403. Josef Sal. History: Measles, pneumonia. Pains began Dec. 6th, 10 a. m.; birth 12:45 p. m. Girl. Length 50 cm., weight 2780 gr. Spontaneous birth. Reflexes 7 days after birth.

No. 3464. H. St. History: Grippe. Pains began Dec. 5th, 6:15 a. m.; birth 7 p. m. Girl. Length 46 cm., weight 2500 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3470. M. Ke. History: Measles, grippe. Pains began Dec. 16th, 9:30 p. m.; birth 11:15 p. m. Girl. Length 48 cm., weight 2700 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3486. Fr. R. History: For four weeks pleurisy. Pains began Dec. 8th, 12 o'clock midnight; birth 1:30 a. m. Girl. Length 47½ cm., weight 2700 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3487. J. G. History: Pains began Dec. 7th, 2 a. m.; birth 11:45 p. m. Boy. Length 47 cm., weight 2450 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3501. An. Sch. Pains began Dec. 7th, 11 p. m.; birth 12:30. Boy. Length 45 cm., weight 2130 gr. Spontaneous birth. Reflexes 9 days after birth.

No. 3503. An. F. Pains began Dec. 7th, 7 p. m.; birth 9:15 a. m. Girl. Length 45 cm., weight 2260 gr. Spontaneous birth. Reflexes 9 days after birth.

No. 3509. Kat. Val. History: Measles. Pains began Dec. 8th, 11:30 p. m.; birth 2:30 a. m. Girl. Length 45 cm., weight 2680 gr. Spontaneous birth. Reflexes 12 days after birth.

No. 3511. An. Auf. History: Measles, asthma. Pains began Dec. 9th, 6 a. m.; birth 11 p. m. Length 48 cm., weight 2730 gr. Spontaneous birth. Reflexes 13 days after birth.

No. 3520. Ant. Fis. Pains began Dec. 9th, 3 a. m.; birth 1 p. m. Boy. Length 49 cm., weight 2800 gr. Spontaneous birth. Reflexes 19 days after birth.

No. 3523. T. Par. History: Bronchitis, grip. Pains began Dec. 10th, 3 a. m.; birth 4:45. Girl. Length 44 cm., weight 2000 gr. Spontaneous birth. Left the clinic on 8th day without any reflex having appeared.

No. 3531. P. L. Pains began Dec. 10th, 1 p. m.; birth 4:30 p. m. Girl. Length 46 cm., weight 2320 gr. Spontaneous birth. Left clinic on 8th day without reflex having made its appearance.

No. 3538. M. W. History. Abortions 1919. Pains began Dec. 12th, 2:40 a. m.; birth 1:15 p. m. Girl. Length 46 cm. Weight 2650 gr. Spontaneous birth. Reflex appeared on the 1st day after birth and after that disappeared.

No. 3545. V. P. History: Typhus. Pains began Dec. 12th, 11 a. m.; birth 1:30 p. m. Length 41 cm., weight 1850 gr. Spontaneous birth. Appearance of reflexes on 9th day after birth and again disappeared.

In order to understand why the hearing perception is absent in the perfectly healthy newborn we are obliged to fall back upon the functional conditions which have been indicated. For as we have mentioned before, every normal birth is associated with some degree of asphyxia in the infant, whereby a sudden change comes in the circulation of the newborn, due to admission of oxygen. All of these conditions can be taken together to explain the decreased hearing ability of the brain in the newborn.

CONCLUSIONS.

My investigations therefore lead me to the following conclusions:

1. The cochlea-palpebral reflex usually appears between the 4th and 6th hour after birth.

2. In premature births, say 6 to 8 lunar months, there is marked delay in the appearance of the reflex, as a rule, first between the 6th and 8th day of extrauterine life.

However, there is sometimes an exception to this rule in that the reflex occasionally appears earlier than this (15 hours after birth) and sometimes later (12 to 19 days after birth).

3. In the hereditary syphilitic newborn, the reflex is sometimes normal and sometimes delayed.

4. Very seldom (2 cases out of the 105 observed) the reflex in those perfectly healthy and of normal birth is sometimes delayed as late as 20 days.

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PRECAUTIONS IN USING LOCAL ANESTHESIA—
REPORT OF A DEATH.

By CURTIS C. EVES, M. D.,

PHILADELPHIA.

The study of the susceptible patient and the method of administration present the most interesting and complex problems in the use of local anesthetics. For the average individual the moderate dosage of a recognized local anesthesia has little danger. It should not be overlooked, in the daily rush of our routine, that even the mildest of these drugs are highly toxic and that there are more than a few individuals who possess an idiosyncrasy for them.

Inasmuch as practically all drugs used for local anesthesia produce almost identical toxic symptoms, novocain or procain, the least toxic of those in general use, will be referred to unless otherwise mentioned.

After the consideration of the hypersusceptible patient the next most important question is the method of administration.

Susceptibility.—Of all the drugs to which a patient may think he is susceptible, his statements in reference to cocain and allied drugs should be considered most reliable. Individuals who have been poisoned by cocain or procain are more susceptible to the second administration. If no history of having had the drug used before is obtained, susceptibility of the patient should always be tested by using a small, well diluted dose previous to giving the full amount necessary for anesthesia. For example, when procain is to be injected for the removal of tonsils, it is my practice to inject about $\frac{1}{2}$ c. c. of a $\frac{1}{4}$ of 1 per cent solution at a point to be used later; after waiting five minutes, if no symptoms are produced, to proceed with the full amount. Similar precautions should be taken before any injection or before applying large amounts of strong solutions of cocain to the mucous membrane.

Administration.—The guiding fact should be first to keep well within the limits of safety in the amount of the drug

used. The dose of procain or novocain as given by different authors varies widely. Roth,¹ after quoting from twenty-one authors, states: "From the above references no definite conclusions can be drawn as to the exact toxicity of novocain in man. Under certain conditions 0.02 gm. (approximately $\frac{1}{2}$ of a gr.) has caused poisoning symptoms, while as much as 2 gr. (approximately 15 grains) has been given without producing symptoms." He further states: "The untoward results that have been reported in the literature from the use of novocain in operations about the head and face might well be accounted for by the fact that absorption may be rapid." "Individual susceptibility is marked in both animals and man."

The dose of cocain has also wide limitations according to the authorities; $\frac{1}{2}$ to $\frac{3}{4}$ grain is usually considered safe.

To keep well within the safety limit, 2 to 3 grains of procain and $\frac{1}{2}$ to $\frac{3}{4}$ grain of cocain can be used in nose and throat surgery.

In the susceptible patient much smaller doses may cause serious toxemia, while in the nonsusceptible individual large doses are safe if guarded by measures to reduce the rapidity of absorption. This can best be accomplished by injecting or applying the drug in only small quantities at a time combined with adrenalin solution or allied drugs which cause vasoconstriction.

Allen² in his book on Local Anesthesia states: "It is believed that cocain once fixed by the body cells (in combinations with them) is not liberated from these combinations as cocain, but as constituent products, ecgonin, etc.; Glaser believes he has isolated ecgonin from the urine. These derivatives of cocain are slightly anesthetic and slightly toxic but much less so than cocain. As a result of the preceding statements it can be said that cocain exerts its full anesthetic or toxic action but once, and if exhausted locally there will be no constitutional reaction."

For injective anesthesia, the smallest amount of the least toxic drug which produces the desired result should be injected slowly, changing the position of the point of the needle after each few drops are injected, thus avoiding the danger of inducing any great amount directly into the circulation should the needle at any time enter a vessel. Owing to the great

vascularity of the tissue surrounding the tonsil. massive single doses injected in one place for anesthesia should be condemned, as should also large cotton tampons hypersaturated with a strong solution of cocain placed in the nasal cavity and left there for a long time. In the applications to the nose and throat precaution should be used to guard against the dropping or running of the solution to other parts.

One should be constantly on watch for the first toxic symptoms, and, since they often occur suddenly and when least expected, should guard himself by the knowledge of how best to combat them.

The first toxic symptoms of cocain are usually those of excitation—talkativeness, laughing, nervous fear, etc. This stage of excitation may be absent, and I believe usually is, when procain or novocain has been used. Nausea, pallor, sweating, pain in the head, faintness and collapse, combined with rapid pulse and a feeling of constriction of the chest, may be seen as early symptoms.

It is a mistake to attribute such symptoms to the nervousness of the patient. They may suddenly terminate in convulsions and death. In severe toxemia, convulsions simulate epilepsy. Beginning with the head and neck, followed by general tremor, short, jerky spasms involve the entire body, are accompanied with cyanosis, tightly clenched teeth with frothy mucus exuding from the mouth, rapid weak pulse, and rapid labored respirations—the pupils may or may not be dilated. The convulsion lasts for a few minutes, but after subsiding for a short time may be followed by others and death.

Treatment.—If the patient is not already in the reclining position he should be placed so at once. Should the state of collapse continue heart stimulants should be administered hyperdermically and inhalations of amyl nitrate or ammonia given. For the convulsive movements, although not tried by the author, inhalations of ether would seem to be by far the best. Artificial respiration and massage of the heart should be practiced.

Quoting from Allen:³ "For nervous excitement or convulsions, H. C. Wood recommends chloroform by inhalations, but it would appear that ether should be better, particularly if it be proved that the central action is associated with the

same vasoconstriction and anemia that takes place in its local field of action; inhalations of ether, due to the tremendous congestion which it produces in these parts, should prove of great value, besides stimulating vasomotors, heart and respiration and controlling the convulsions; nitrous oxid would be equally as valuable; amyl nitrate, while producing the same congesting effect, would not exert the same controlling influence upon the convulsions.

Engstady⁴ says, in speaking of cocain poisoning: "In the first few cases I was called on to treat, strychnin and morphin in combination were used with marked benefit. But, as cases kept multiplying, I found the action of these drugs too slow, and I decided that there must be something to counteract the poison more rapidly when life was in extreme danger. It was necessary to find a remedy that could be administered at any time and be instantaneous in its action. I soon found ether to be the required drug. This was administered as ordinarily given to produce surgical narcosis. Ether stimulates the vasomotor system, is a tonic to the heart muscles, stimulates the action of the respiratory centers of the brain and of the pneumogastric nerve, and increases the pulmonary circulation in the first stages. While cocain inhibits the action of the heart, especially on the right side, it has also a marked inhibitory action on the respiratory centers of the brain. Death may occur from feeble respiratory movements of the so-called Cheyne-Stokes type or asphyxia.

"To me ether has proved extremely valuable. It has saved what seemed hopeless cases. It stimulates the heart and the respiratory system almost instantly. The pulse becomes fuller at once and of normal tension. The marked mental excitement is allayed as the poison rapidly disappears. The individual regains consciousness as soon as the effect of the small amount of ether has disappeared."

Allen⁵ further advises the use of opium and its alkaloids. He believes, however, it is best used as a prophylactic or preventive by the preliminary use before the injection of the anesthetic "when it does certainly permit the use of much larger doses of the anesthetic and in this way acts as a direct antagonist."

Allen⁶ does not think the injections of adrenalin advisable after the toxic symptoms. He believes they are caused by paralysis of the higher nerve centers, adrenalin exerting the same action upon these centers as it does locally, causes a prolonged retention of the toxins in the brain.

On the other hand, Eggleston and Hatcher⁷ and Braun⁸ advise the infusion of epinephrin solution upon the recognition of the first toxic symptoms to stimulate the action of the heart so that the toxic agent may be brought in contact with the liver, where Eggleston and Hatcher believe the toxins are destroyed, quoting from Eggleston and Hatcher:⁷ "Since acute poisoning in man occurs where least expected, those who frequently employ local anesthetics should always be prepared to apply the three resuscitative measures of intervenous infusion of epinephrin, artificial respiration and cardiac massage. All three should be used in combination, since no one is efficient, except in rare instances." They base their belief upon extensive experiments upon cats; they were able to resuscitate cats after a rapid intervenous injection of doses of local anesthetics up to twice the usual fatal dose by using the above measures.

All recent articles written upon this subject claim that these drugs kill by paralysis of the heart and respiratory centers.

The committee appointed by the Otolaryngologic Section of the A. M. A., to study the "Advantages and Disadvantages of Various Local Anesthetics in Nose and Throat Work,"⁸ after two years' study and communication with the members of the otolaryngologic section, report forty-seven deaths from local anesthesia; all except eight cases were tonsil cases. Of the cocain deaths it is appalling to note that one-fourth were due to "mistakes of druggist or nurse."

After carefully selecting the tonsil cases which had no contributing causes, such as tuberculosis, heart disease, mistakes, etc., reported, I found nine deaths caused from injections of cocain ranging in strength from 0.2 per cent to 3 per cent. Twelve deaths resulted from injections of procain solutions ranging in strength from 0.5 per cent to 2 per cent. In thirteen of these epinephrin or adrenalin was used in the solution. In the procain cases eight had concain applied to the pharynx.

gical surface before injection. Four had only procain injections.

The greater number of procain deaths in these selected cases occurred because of better reports given of them. In the full number of forty-seven, twenty-five were cocain deaths, fourteen were procain deaths. The other deaths were: One, from the use of alypin, which is very toxic, and two from apothesis. Morphin injections had been given to approximately half of the cases. Procain or cocain was the only constant medication.

Becker¹⁰ reports a death of a girl 25 years old following an injection of about 100 minims of 1 per cent novocain for the removal of tonsils. The patient died suddenly with symptoms simulating an epileptic convulsion. A larger amount of solution from the same container had been used upon a sister in the same manner without any toxic symptoms.

Simpson¹¹ mentions a death of a male patient before a tonsil operation had been begun. Hypodermic morphin, gr. 1/6, was given one-half hour before, followed by local application of 10 per cent cocain solution fifteen minutes before. While in the act of injecting $\frac{1}{2}$ of 1 per cent novocain the patient was suddenly attacked with what Dr. Simpson thought was an epileptic seizure, followed in a short succession by six or eight more convulsions, and died shortly after.

White¹² reports a tonsil case which was being prepared for operation in his clinic. "The patient, a woman, aged 23, rather heavy set, was prepared for local tonsillectomy without the previous use of morphin as follows: The tonsils, pillars and pharynx were swabbed with a 20 per cent solution of cocain with 10 to 15 drops of 1/1,000 adrenalin to the ounce. This was followed in about ten minutes by the injection of a 1 per cent solution of novocain. About three drams of the solution was drawn up into the ordinary type of syringe used for this purpose, and the injection was made in the usual sites on the right side and about the same amount was injected on the left side. About fifteen seconds after the last injection the patient with an exclamation reeled forward in convulsions. Cyanosis was marked. The patient was laid flat on her back on the floor, the sphincter ani dilated and artificial respiration begun, but the convulsions persisted and cyanosis was still very marked; the pupils were semidilated, the pulse very faint, and

the respirations shallow. The heart was massaged, ether and adrenalin given subcutaneously, and the use of the pulmotor was instituted. The convulsions ceased, no respiration was evident and there was no perceptible heart beat or pulse. The artificial respiration was continued for about an hour, at the end of which time the patient was pronounced dead. There was no apparent cause for the condition. At the postmortem all the organs were found normal, excepting the thymus, which weighed about 50 grammes. Diagnosis: Status lymphaticus."

After operating upon nearly 1,000 tonsil cases under local anesthesia without any serious symptoms, a patient I was carefully preparing for operation before a public clinic suddenly died before the operation had been begun. On March 15, 1921, R. A., a young man, 18 years old, blond, of medium build, with rather effeminate features, was prepared for operation as follows: Morphin sulph., gr. 1.4, with atrophin sulph., gr. 1/150, had been given twenty minutes previously. Ten per cent solution of cocain was applied to the superior and inferior poll of each tonsil by placing a cotton tipped applicator saturated with the solution for a few seconds in each place.

Following this about 1½ drams of 1 per cent procain solution containing 1 drop of adrenalin, solution 1/1,000 to the dram (approximately 1 1/5 grains of procain plus 2 drops of adrenalin solution), was injected outside of the capsule on each side. In about one minute after the last injection, while waiting for the anesthesia to take effect, the patient said he felt badly and looked very pale. His head was placed between his knees for perhaps another minute, when he began with a slight convulsive movement involving the head and upper part of the body. He was at once placed upon a stretcher, by which time the convulsive movements involved the whole body. Short, rapid, jerky spasm, lifting the extended legs and head and shoulders from the stretcher, was accompanied by a general tremor, marked cyanosis, eyes open, tightly clenched teeth, with biting of the tongue and frothy bloody mucus exuding from the mouth. The pulse was weak and rapid, becoming almost imperceptible.

The respirations were shallow and labored. After lasting one or two minutes the convulsions subsided to a general tremor, with improvement of the pulse.

A second convulsion followed, after which the patient was taken to the ward, where the pulmotor and other measures were used to resuscitate him without avail. He died in about thirty minutes.

Solution from the same bottle had been used for a patient just previous to this one without toxic symptoms.

Having been told before the clinic began that one of the patients to be operated upon was subject to epilepsy, I at first mistook the symptoms for an epileptic seizure.

The autopsy showed a much enlarged spleen and general hypertrophy of the lymphoid tissue throughout the body. The postmortem diagnosis was "status lymphaticus." The fact remains that the patient died with symptoms of novocain poisoning.

To almost every laryngologist there comes a period of enthusiasm for the use of local anesthesia in tonsillectomy. To operate upon a conscious patient without pain or bleeding and to have a grateful patient, grateful especially because of the elimination of the fear of ether and its after effects, is encouraging; add to this a more or less lengthy series of such cases, anesthetized with a comparatively small amount of a solution of less strength than the general surgeon uses in almost limitless quantities—and the enthusiasm of the young laryngologist is apt to relax his vigilance and to cause him to select too wide a field for the use of local anesthesia.

It needs to be constantly borne in mind that ether, except in selected cases, is the safer anesthesia.

There is a large number of selected cases in which local anesthesia is the method of choice. This is especially true in operations upon the nasal cavity, in pulmonary, renal, hepatic disease and in the aged.

Local anesthesia is especially contraindicated when operating upon the nose or throat of children, highly nervous or neurotic subjects, in epileptic and static lymphatic cases and in some cardiac conditions.

SUMMARIZING THE PRECAUTIONS IN USING LOCAL ANESTHESIA.

1. A careful history and physical examination of the patient, especially to detect susceptibility, neurosis, epilepsy, cardiac lesions and the existence of a lymphoid diathesis.

2. Elimination of the patient's susceptibility to the drug used.

3. The use of the smallest amount of the least toxic drug well diluted which will produce anesthesia.

4. The addition of adrenalin or epinephrin to all solutions to reduce the rapidity of absorption.

5. The solutions should always be administered slowly. When injections are given every care should be taken not to enter a vessel.

6. The reclining position is to be preferred, thus relieving all strain from the heart.

7. It should be the infallible rule of every surgeon to freshly mix his own solutions and to keep the stronger solutions for surface application in a different colored container.

8. The operator should be prepared at all times to administer the measures to combat toxic symptoms.

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LXVIII.

THE WARING SUCTION TONSILLECTOMY.

BY J. B. H. WARING, M. D.,

BLANCHESTER, OHIO.

In recent years, especially perhaps in the past decade, the tonsil question has loomed larger and larger to the medical profession, and in common with the laity, to a large extent has arisen the idea that the tonsil affords the chief gateway for entrance of various systemic infections. Increased knowledge of pathology in general, and especially the development of the focal infection theory, have served to place the tonsil in the medical limelight more and more.

Tonsillectomy has become a very common operative procedure with the growing demand for removal of tonsils in adults afflicted with this and that ailment. Conservatism is in the air, however, and it is needless to caution against indiscriminate removal of the tonsils, and to emphasize that tonsillectomy should not be resorted to until all other likely etiologic factors have been eliminated in the given case.

I think it is universally admitted that complete removal of the tonsil, including the capsule, the so-called radical tonsil operation, is the operation of choice on the part of the great majority of American rhinologists, in the belief that unless the entire tonsil is removed full benefits may not be secured.

It is true that some rhinologists do not favor removal of the tonsillar capsule, but this view is not held by the majority, and removal of the capsule certainly better insures total ablation of the tonsil and greatly facilitates operative technic.

It is unfortunate that there has survived from the older "clipping," or partial removal tonsillectomy, a rather widespread impression that there is little danger in removal of tonsils, and that it is an operation to be performed with impunity anywhere and by any physician. We should strive to correct this impression and have it more generally realized both by the profession and by the laity that tonsillectomy is more of a major surgical procedure, not unattended with danger, under

even the most favorable conditions, and an operation to be performed as far as possible in hospital or well equipped office only and by trained rhinologists.

As the case stands today, however, tonsillectomy is performed not only by the trained throat specialist, but almost as commonly by the general surgeon; by the general practitioner with surgical leanings, and also by the recent graduate ambitious to "rise in his profession." Tonsillectomy today is performed in hospital, in office and in the home; with assistants and without; under general anesthesia and under local anesthesia.

Tonsillectomy technics today may roughly be divided into two main types, namely, some form of snare and dissection and some form or modification of the ingenious method brought out by Sluder in 1910.

The innumerable variations of this and that technic, together with the multitude of tonsillotomes, tongue depressors, retractors, tonsil hooks and tenaculums, knives, scissors, punches—all serve to indicate that the last word has not been reached in tonsillectomy technic; that the methods in use today offer certain dissatisfactions in end-result or difficulties in operation which lead the earnest operator of mechanical turn to strive to improve his armamentarium and his technic.

Some form of snare and dissection is perhaps most used today, and in the hands of experts and on tonsils of appropriate size and type gives very satisfactory result. In the hands of the average operator, however, snare and dissection technics usually leave considerable to be desired. The operation is often tedious and time consuming, and a strain on the patience both of the patient and operator under local; and under general anesthesia often means much time lost under anesthesia. Dissection work is often bloody and, with a field obscured by blood and mucus, most difficult and trying. Advent of suction pump apparatus has been a great advantage in clearance of the operative field, but even with this, dissection work on badly adherent and submerged tonsils often degenerates into a mere cutting out procedure instead of orderly dissection. There is often more or less damage to pillars and laceration of throat muscles, so that convalescence is often

delayed, stormy and painful. On friable and ragged tonsils it is often difficult or impossible to obtain a satisfactory grasp with any of the numerous tonsil seizing forceps, screws or tenaculums; or when a satisfactory grasp is obtained most annoying and delaying to have the instrument pull out of the tonsil at a critical stage, with attendant shredding of the remaining tonsil tissue. More or less free bleeding is often met with in dissection work, and aside from the weakening effect of loss of blood, much valuable time is often lost in efforts at hemostasis. The extensive armamentarium usually at hand for this type of tonsil operation likewise has a rather disconcerting effect when viewed by the patient under local. Often again snare and dissection means repeated snaring of bits of tonsil tissue left—more labor and more time consumed.

Turning to the ingenious technic of Sluder with its various modifications, a few of its disadvantages may be pointed out briefly in passing. Advantages claimed for it are rapidity, minimum of surgical violence and less hemorrhage.

In the hands of experts in its use, the Sluder type operation is almost spectacular in its comparative rapidity and clean end result, but it is not a technic applicable to all forms of tonsils, and the Sluder operator soon learns to recognize the tonsils he cannot "get" satisfactorily with this technic, and turns to some other secondary mode of attack. Usually the tonsils hard to "get" with the Sluder technic are the small, deeply submerged and adherent tonsils, so often badly diseased and in need of removal. Quite often one or both pillars are damaged or even completely removed by the Sluder method, and with the so-called sharp Sluder free hemorrhage often follows the enucleation. With the dull or two bladed type of Sluder instrument, such as the LaForce, Beck, Meding, etc., a practically bloodless enucleation may be done, and on appropriate type tonsils a very satisfactory operation. However, the instrumental manipulation called for by this technic is often quite difficult for the average man to acquire satisfactorily, and its successful performance calls for a considerable degree of "strong arm" muscular accompaniment, which all operators do not seem to possess. Likewise visibility at certain stages of the manipulation is rather obstructed, with instrument and fingers in mouth and throat at same time.

It has been my intention to point out briefly in passing such disadvantages of the two main type tonsillectomy operations in use today as might appear to the average operator and not to decry in any way the hundreds of brilliant operative results secured by expert operators every day by these two technics.

It has been with an open mind, but faced with the difficulties and disadvantages of modern tonsil work, that the subject of tonsillectomy has been approached by the writer from what may be termed a new or a modified angle, in an effort to perfect a tonsillectomy technic which would be equally applicable to all types of tonsils; be rapid, clean and bloodless, and likewise insure good tonsil work in the hands of the beginner and the occasional operator, no less than in the hands of the average and the expert operator.

In a considerable use of tonsil suction tubes for diagnostic and therapeutic purposes, I was impressed not only with the thorough mechanical "vacuum cleaner" cleansing of tonsils by suction, but equally so with the ease and completeness with which even deeply submerged tonsils were drawn out of their beds and held firmly in the mouth of the glass suction tube while the suction pump was turned on.

Pus and masses of caseous and ill smelling purulent exudate were drawn out of the tonsil crypts and into the suction tube, thus mechanically removing from the tonsil and from the throat much potentially infective material, which if left and swallowed or reabsorbed could well cause considerable trouble.

In the opinion of the writer, this "vacuum cleaner" mechanical cleansing of the tonsils prior to actual operative attack can well be incorporated as a preliminary step in tonsillectomy technic, applicable to any and to all tonsil removal operations.

As one confrere wrote me recently: "It occurred to me how easy it would be to throw a snare loop over the tonsil already almost turned inside out by grasp of the suction tube and thus enucleate." Using an appropriate size glass suction tube, it was found that the tonsils were drawn out of their beds and held in the mouth of the suction tubes far better than with any of the numerous tonsil grasping forceps, and held there practically inverted as long as suction was on. Even without anesthesia of any sort patients complained of but little dis-

comfort during the maneuver, and there was no laceration or shredding of tonsil tissue as forceps so often entail. As desired, however, the suction grasp on the tonsil could be instantly released at will by disconnection of the suction pump and as easily regrasped by suction.

High visibility was another feature that appealed to me. One can see exactly what he is doing, as the clear glass of the suction tube offers little obstruction to vision.

With the first few enucleations attempted with the snare and suction tube, a plain glass tonsil suction tube of the Hurd type was employed, but it was found this was not satisfactory, because in a certain percentage of cases the snare loop would hang up on the lip of the tube and as tightened down slide down towards the shaft of the tube instead of on to the base of the tonsil to be enucleated. This necessitated release of the tonsil, readjustment of snare and a fresh start. In the cases where the snare loop did slide down over the base of the tonsil it was found enucleation could be performed rapidly, cleanly and in a practically bloodless manner. The tonsil was removed cleanly in capsule, with no damage to pillars, uvula or other throat structures; the fossæ left clean and convalescence rapid and uneventful.

To overcome the only mechanical imperfection met in the procedure I have designed a new type of tonsil suction tube, all glass with rubber handle for ease of manipulation, and in a set of varying sizes to accommodate all sizes and types of tonsil. These tubes have a rather bulbous head set at about right angles to the shaft of the tube, short necked and with smooth shoulders sloping rapidly down to the aperture, which is broadly oval in its perpendicular axis.

With these improved suction tubes the operation may be said to be 100 per cent perfect in execution and in end result. Practically all physicians doing throat work at all have some form of compressed air and suction apparatus in use, and any of the modern suction pump apparatus should give sufficient suction for this operation.

Where general anesthesia is employed, a small vacuum bottle of from $\frac{1}{2}$ to 1 pint capacity may be incorporated in the rubber tubing vacuum circuit back to suction pump, so that as needed the throat may be cleansed of mucus and saliva,

using the same glass suction tube as for enucleation, but merely turning same with aperture down into the pharynx. Owing to its rapidity and bloodless type of operation, only a very brief and light general anesthesia is required, as little or no time under anesthesia is expended in efforts at hemostasis.

Where local anesthesia is employed, the tonsillar region and pharynx may be anesthetized by whatever method the individual operator prefers. The operation is essentially a one-man job and may be employed equally well in hospital or office and, except for an anesthetist where general anesthesia is employed, no other assistance is required in the operation.

Any type of tonsil snare may be employed, although in the opinion of the writer snares having a ratchet mechanism, such as on the Beck-Pierce and similar snares, offer certain advantages in a more nearly bloodless enucleation. About a No. 8 snare wire gives good results. As a rule the snare and suction tube are the only instruments required, the suction tube being used somewhat as a tongue depressor as it is carried into the mouth and over the tonsil to be enucleated.

An appropriate sized Waring suction tube having been connected up to the suction pump by about four feet of flexible rubber tubing, the snare loop is now passed over the suction tube and allowed to hang freely suspended out of the way until required. The suction tube should be of a size sufficient to firmly grasp and encircle the tonsil to be removed. Under direct inspection the suction tube is carried into the mouth and its mouth carried between the pillars and over the tonsil to be removed. Suction is now turned on, when it will be seen through the clear glass of the suction tube that the tonsil is drawn completely out of its bed, almost inverted, and firmly held in the grasp of the suction tube. High visibility is one of the most appealing features of the operation to me; at every stage you can see exactly what you are doing. There is no danger of including the uvula or damaging the pillars by a grope in the dark. Likewise success of the operation does not rest on heavy muscular exertion or difficult instrumental manipulation in the throat; the operation is simplicity itself, as will be seen at a glance from the cut. There is no preliminary dissection, no employment of knife, scissors, retractors, tonsil seizing forceps, etc.

As the tonsil is grasped by suction the snare loop is now carried over the bulbous end of the suction tube, and on to the base of the tonsil, the snare being grasped by the left hand for the right tonsil and by the right hand for the left tonsil. As the snare loop is tightened down, the smooth, sloping shoulder of the suction tube automatically carries it down and over the base of the tonsil to be enucleated. At this stage the tonsil may be enucleated rapidly; or, if desired, enucleated slowly by a gradual screwing down of the snare loop, as is possible with the Beck and similar type snares. This gives a more bloodless operation, but is not essential. The operation will be found practically bloodless in all cases, the rather heavy snare wire crushing rather than cutting through tonsillar vessels as the base of the tonsil is separated from its bed.

It will be found the tonsil is practically inverted and cleanly enucleated in capsule, fossæ left smooth and no damage to pillars or other throat structures. There is no cutting or laceration of the pharyngeal muscles, as often occurs in dissection work, and as a result a minimum of postoperative sore throat is complained of, and convalescence very rapid and uneventful; the majority of patients eat their regular meals the second morning. Owing to its rapidity and bloodless nature there is practically little shock, even in patients comparatively advanced in years.

The second tonsil is removed in the same manner. Any oozing in fossæ may be very effectually controlled by a swab of Monsel's solution in glycerin, but this is rarely needed, and secondary hemorrhage after this technic has not been encountered in any case.

Advantages of the suction tonsillectomy over preexisting technics are briefly as follows:

The operation is so simple that even the beginner and occasional operator can do good, clean, bloodless tonsil work with the assurance of uniformly good end results, and do it in a manner more rapid and more satisfying both to patient and to operator than with the older technics.

The operation is practically bloodless in all cases, so that no time is lost in efforts at hemostasis. Where general anesthesia is employed, its rapidity and clean type calls for only a very brief and light anesthesia, so no time is lost in hemostasis.

The operation is essentially a one man job and does not require the train of assistants and extensive instrumental armamentarium usually met with in tonsil work.

High visibility is another strong feature of the operation. Every step of the operation is under direct inspection, and there is no difficult instrumental manipulation inside the mouth as with Sluder type operations. The technic is simplicity itself.

There is no type of tonsil the operator cannot "get" easily, rapidly and cleanly by this operation, and its superiority will quickly be demonstrated on the small, deeply submerged and adherent tonsils so difficult of clean attack under ordinary methods.

On account of its clean type, smooth fossæ and lack of damage to adjacent throat structures, very little postoperative sore throat is experienced and convalescence **will be found** rapid and uneventful.

109 EAST MAIN STREET.

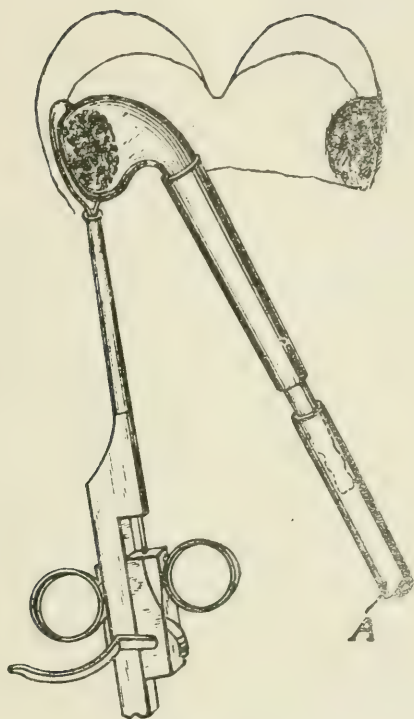


FIGURE 1.

The right tonsil is engaged in the bulbous mouth of a Waring suction tube, which is connected by rubber tubing "A" to suction pump. Snare loop has automatically slipped down over sloping shoulders of suction tube in readiness to enucleate. Beck-Pierce snare in use in illustration, but any type of tonsil snare applicable.

LXIX.

OTOGENIC SYMPATHETIC MENINGITIS—REPORT OF A CASE WITH RECOVERY AFTER A RADICAL MASTOID OPERATION.

BY JESSE WRIGHT DOWNEY, JR., M. D.,
F. A. PACIENZA, M. D., AND OSCAR G. COSTA, M. D.,
HOUSE STAFF, MERCY HOSPITAL,
BALTIMORE.

The following case record is a striking example of the variously called sympathetic, protective or serous meningitis, due to a chronic suppurative otitis media presenting classical symptoms, typical laboratory findings and a surgical demonstrable area of meningeal irritation.

It is undoubtedly of the class in which the great majority of reported recoveries from otogenic suppurative meningitis probably belong, and it substantiates the experiences and opinions of Eagleton, Yerger and others that meningitis of otitic origin may be amenable to surgical treatment so long as it remains in protective stage, which is indicated by a sterile spinal fluid.

Jos. P. White, age 49, was admitted to the Mercy Hospital May 20, 1922. He had been previously treated by Dr. M. Raskin for a chronic suppurative otitis media of twelve years' duration, which had recently increased in severity with periodic manifestations of severe pain, headache, vertigo and unconscious spells, which lasted from three to five minutes and had been called epilepsy. The left ear had shown a large polypoid mass, whose atypical appearance suggested malignancy and justified removal for microscopic examination. A portion of the polyp was carefully removed by Dr. Raskin on May 18th, and within forty-eight hours the man returned almost frenzied with headache, mentally confused, and evidently very ill. He was immediately sent into the hospital, where Dr. F. A. Pacienza, Dr. O. G. Costa and I examined him shortly after his admission, finding the following diagnostic symptoms:

Patient poorly nourished, semiconscious, moaning with pain in head, restless and irritable. Questions indefinitely answered. Temperature, 102. Pulse, 120. Respiration, 26.

Indefinite paresis of left external rectus. Pupils and eye-grounds normal.

Profuse offensive purulent discharge from left ear. Drum absent, tympanum filled with granulations. Left mastoid and parietal regions tender on palpation.

Neck and back muscles rigid; patient showing definite opisthotonos. Kernig's sign positive. Brudzinski's sign positive. Reflexes exaggerated. Slight spontaneous tremor of arms and legs. Heart, lungs and abdomen negative.

There could be but little doubt that we were dealing with a septic meningitis of otitic origin, and this impression was confirmed by a spinal puncture. The spinal fluid was under marked pressure, creamy in appearance, and the following laboratory report was given: Cells, 6,000 to cu. m., 96 per cent polymorphonuclears, 4 per cent lymphocytes. Globulin positive. Wassermann negative. No microorganisms found in stained slides made from a centrifugalized specimen.

The blood count showed a leucocytosis of 19,900, with 92 per cent polymorphonuclears, 5 per cent small mononuclears and 3 per cent mononuclear cells.

The absence of bacteria in the spinal fluid, in conjunction with a chronic suppurative otitis media, now exhibiting the signs of an acute exacerbation, made me feel that an exploratory radical mastoid exenteration was justifiable. It was impossible, however, to get the consent of the patient's family until his wife, who was out of the city, could arrive. This meant a delay of another twenty-four hours. In the meanwhile the man's condition remained unchanged and cultures made on blood agar from the spinal fluid remained sterile. This laboratory report almost assured us that we were dealing with a so-called protective meningitis, and the chances of success from a surgical procedure were, therefore, somewhat better. Accordingly on May 22d, a mastoidotympanal exenteration was performed.

The cortex was extremely hard, but the cellular structure of the bone was not entirely sclerosed and a softened spot was found in the upper posterior region of the mastoid which, when curetted away, disclosed an absence of the bony wall of the sinus over an area at least a centimeter in diameter. The edges of this opening were smooth and the surrounding bone

sclerotic, making it evident that the bone destruction was of long standing.

The exposed dura was covered with small vascular granulations and was bound by adhesions around the whole circumference of the exposed area. We were very careful to avoid removing or breaking down any of these dural granulations or adhesions.

The mastoid antrum was deep, of average size, and filled with pus, granulations and cholesteatomatous material, with an odor indescribably offensive. The dura was not found exposed in this region and the roof of the antrum and tympanum appeared healthy.

The plastic part of the operation was omitted and the wound left open and packed with iodoform gauze.

Cultures made from the mastoid at the time of operation remained sterile. It is interesting to note, however, that these cultures were made from the region of the dural exposure and not from the foul secretion found in the antrum.

On May 23rd, the patient's mental condition showed marked improvement. He was more comfortable and answered questions fairly accurately. Maximum temperature, 101; pulse, 100.

On May 24th, his condition was markedly improved. Temperature, 99; pulse, 90. Very little headache. A spinal puncture was made, which showed the fluid under but slight pressure and but faintly cloudy. A count of the cells showed 90 to the cu. mm., and the differential count showed polymorphonuclears 8.5 per cent, small mononuclears 86.5 per cent, large mononuclears 5 per cent. The wound was draining freely and very foul.

On May 26th, the temperature was normal. Another spinal puncture gave a clear fluid under no pressure. Globulin still positive.

On May 28th, spinal puncture, fluid clear, no pressure, globulin negative, and no cells.

On June 26th, the man was discharged from the hospital, mentally clear, and in good physical condition.

When last seen (October 9, 1922) he was back at work, apparently in good health, free of headache and his so-called epileptic attacks.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, March 5, 1923.

THE PRESIDENT, DR. C. H. LONG, IN THE CHAIR.

Paper: "Parotid Fistulæ in Mastoid Operations."*

BY WILLIAM E. GROVE, M. D.

MILWAUKEE.

DISCUSSION.

DR. JOSEPH C. BECK said he never had encountered such an unfortunate condition as a double fistula of the parotid gland. He did not think it likely that the Doctor's case was the result of injury by a sharp instrument on both sides. The only experience Dr. Beck had had with a parotid fistula was where a nerve anastomosis between the facial and spinal accessory nerves was performed. In that specific case the assistant penetrated the parotid gland with a sharp retractor, and Dr. Beck called his attention to the probability of a parotid fistula resulting from that accident. It did, and continued to discharge for several weeks. The introduction of a conical point which was charged with electric heat (surgical diathermia) finally succeeded in closing the fistula. No doubt this procedure resulted in the coagulation of the epithelium lining the fistula and allowed cicatrization to take place. This case was reported in connection with a few cases that were operated on for anastomosis. The facial paralysis, however, did not recover.

DR. J. HOLINGER reported an experience with a parotid fistula in the three-year-old girl of a colleague. The abscess of the parotid started one week after operation. The parotid was not injured during the operation. In order to avoid a scar on the face later he incised the abscess subcutaneously from the mastoid wound. He advised the parents to omit all fruit from the diet, but the father thought this was a "grandmother

*See page 855.

fad" and paid no attention to it. The abscess persisted and necessitated daily changes of dressings for six weeks. Dr. Holinger was convinced that the fruit acid caused an increase in the secretion of saliva and persuaded the father to leave off all fruit and fruit juices, whereupon the fistula closed in two days.

DR. GEORGE E. SHAMBAUGH said that study of the anatomy of the external meatus has disclosed a condition which permits of extension of infection from the parotid into the external meatus and of an infection in the walls of the canal through to the parotid. The anterior wall of the external part of the canal is formed by a wedge shaped tongue of cartilage which has two clefts called the incisura of Santorini. These clefts are necessary for the movement of the auricle in the lower animals. It is through these incisura that the infection extends. Dr. Shambaugh had never seen a case where a furuncle had broken through the meatus into the parotid, but he had seen an abscess of the parotid which broke through into the external meatus.

DR. OTTO STEIN said he had had no experience with parotid fistulæ in connection with mastoids. He had seen an abscess of the parotid break through into the canal followed by a fistula and had seen one case of fistula in the parotid canal due to infection from opening a furuncle, but he had never seen such a case as that reported by Dr. Grove.

DR. W. E. GROVE (closing) said that when the first of the fistulæ occurred he was willing to plead guilty to having been clumsy in his operative procedures and possibly to having injured the parotid gland in some way, but when he operated on the child on the opposite side with the first experience in mind, he was certain he did not injure the parotid gland. His only explanation for the thing occurring on both sides was that there must have been a much displaced parotid gland which projected backward into the field of operation.

He agreed with Dr. Holinger that with a salivary fistula of this kind it would be wise to avoid the use of any foods that would stimulate the flow of salivary secretion. The more a fistula can be kept from discharging the better chance there is for prompt healing.

Paper: "Experiments on Overirritation of the Labyrinth (at Professor Siebenmann's Clinic of Basel, Switzerland)."

By J. HOLINGER, M. D.

ABSTRACT.

The essayist briefly reviewed a long series of experiments that have been published under several names in the *Zeitschr. f. Ohren.*, and the special work by N. Satah, and presented lantern slides which demonstrated this interesting work in detail.

He stated that the middle ear was always found free from changes if there had not been any intentional interference, such as removal of the anvil. The vestibulum and the semicircular canals were also free from change. The cochlear nerve, the spiral ganglion and the acoustic nerve were free in the beginning but became successively atrophic with the progress of the atrophy of the respective parts of the organ of Corti.

These experiments showed that bone conduction does not influence the result of overirritation. In the human there is increased bone conduction in disease of the middle ear, but if this were as important as Wittmaack believes, the ears without the anvil could not show an intact organ of Corti. Hoesslin and Satah concur in the opinion that the membrane of Corti is the active part, and this corresponds with the findings of Dr. Shambaugh in the ampullæ, where a similar organ, the cupola, irritates the hair cells. It is not known how the membrane of Corti is moved.

From these experiments the essayist drew the following conclusions:

1. If animals are exposed to very loud noises for several hours on many successive days, the entire organ of Corti becomes atrophic and finally is displaced by connective tissue in all convolutions of the cochlea.
2. If animals are exposed in a similar manner after removal of the anvil, atrophy does not take place, showing that hearing is seriously interfered with if the middle ear is not intact.
3. If animals are exposed in a similar manner to clear sounds—i. e., sounds without overtones, the atrophy of the organ of Corti is circumscribed. The area of degeneration is located

nearer to the base of the cochlea as the sound which causes the degeneration is pitched higher. The lower in pitch the sound which causes the degeneration, the higher in the cochlea is the area of degeneration.

4. If very loud shots are fired close to the ears of the animals (without any bodily injury except by the noise) the animal becomes unconscious and remains in this state for several hours or even days. If the animal is killed while in that state the hair cells and Deiter's cells are found in complete disorder; some of them appeared as if torn out of their place and laid in different positions, others looked as if depressed or crushed.

5. If animals are examined at different periods after being exposed to the noise of loud shots, the organ of Corti is found in different stages of regeneration. After six weeks the repair is complete but the animal is fretful, psychically altered (shell shock).

6. If the animal is exposed the second time to the noise of a loud shot close to the ear, the same change is found immediately after this exposure, but subsequently no regeneration takes place; the whole organ of Corti is replaced by connective tissue, the animal is completely deaf and the atrophy progresses to the cochlear nerve, the spiral ganglion and the acoustic nerve.

7. In birds the organ of Corti is much wider and flat, not rolled up in the form of a snail, as is the case in higher animals, but similar results were obtained. After the shooting the following facts were observed: The outer parts of the organ of Corti were much more changed than the inner parts. The outer hair cells were crushed and torn out of their position while the inner hair cells were practically intact. This led to the final conclusion that in the act of hearing Corti's membrane does the vibrating and not the basilar membrane. In the shooting experiments the end of the membrane makes the largest excursions and does the greatest damage; near the insertion the excursions are smaller and consequently the inner hair cells remain intact.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH expressed himself as quite in accord with Dr. Holinger that this research was one of the

most interesting as well as the most instructive investigations that has yet been undertaken on the internal ear.

When Wittmaack first published the results of his work Dr. Shambaugh examined the drawings with a great deal of interest, but could not help being somewhat skeptical about interpreting slight changes as being the result of overstimulations. He has so often observed in his own work changes in the organ of Corti which were artefacts. Fixing fluids penetrate the labyrinth very poorly and the delicately constructed organ of Corti soon undergoes postmortem changes.

Dr. Shambaugh has often resorted to the extraction of the stapes in order to permit the more rapid penetration of the fixing fluid. When this is done without tearing the endosteum covering the footplate, a very good result can be obtained. To obtain fixation, however, by the ordinary method of dropping the tissue in the fixing fluid does not always give satisfactory results. Often the organ of Corti in the basal coil was in perfect condition, but postmortem alterations were prevalent throughout the upper coils.

An opportunity came some few years later to examine the preparations made in Siebenmann's laboratory, and there was no room for confusing the actual degenerations with any artefacts. The particular things of interest brought out in this research are that different areas of the organ of Corti undergo degeneration with the different types of whistles used in the stimulation. The higher the pitch the lower down toward the beginning of the basal coil was the degeneration area. It was noted that for each tone used a rather extensive area of the organ of Corti was involved. It would seem that a single tone produces stimulation of rather an extensive area in the Corti's organ. This does not in any way conflict with our conception of a peripheral tone analysis. All that is required to make this possible is that a different group complex of hair cells are stimulated for each tone in the scale.

It was very interesting to note that where the conduction mechanism was broken by the extraction of the incus, this method of overstimulation did not produce the characteristic degenerations in the cochlea. The results of loud explosions were particularly interesting. Where the organ of Corti was examined immediately after the explosion as, for example, the

shot of a pistol close to the ear, one invariably found Corti's organ as though blown to pieces. If, however, the animal was not killed until some weeks later, one of two things was found. Either the organ of Corti had undergone complete degeneration or there had been a restitution of the normal condition. This was exactly what was found so frequently during the war. Some of these cases remain permanently deaf, and others show a partial or even a complete return of hearing later on.

Some of the men who were studying these cases during the first period of the war tried to explain these phenomena on the basis of an injury to the nerve fibers in the brain. Dr. J. Gordon Wilson seemed to be inclined to this view. He thought it was Dr. Holinger who, when Wilson made his first report here, called his attention to this experimental research by Wittmaack and Siebenmann. Later Dr. Wilson accepted the view that the defects in the hearing caused by concussion were the results of peripheral alterations.

Dr. Shambaugh stated that the society should feel grateful to Dr. Holinger for bringing this very interesting study before them.

DR. JOSEPH C. BECK thanked Dr. Holinger for bringing out this work and called attention to the work done by Yoshi, who is really the great man in Tokyo today. He did a great part of this work and never for a moment forgot or neglected to point to Siebenmann as the man who originated the work. He was kind enough to show Dr. Beck how to preserve a specimen from a case of otosclerosis, in the event he got a postmortem where he could get the labyrinth and fix it, and he showed him how to inject the labyrinth. Dr. Beck had found, as others had, the great amount of artefacts when the specimens were removed by the old methods of fixation. Yoshi took an animal (guinea pig) and thrust a trocar into the heart and then by gravity injected the entire head, including the labyrinth. He then removed the temporal bone and showed him how wonderfully the labyrinths were bathed in the injected fluid. Dr. Beck thought this work of Yoshi's was a very valuable contribution.

DR. ROBERT SONNENSCHNAIN thought that two of the outstanding points in the address were, first, the light thrown

upon the function of the tectorial membrane to which, as had been stated, Dr. Shambaugh called attention years ago and was the first to offer a better explanation for the theory of hearing than was given by Helmholtz, who used the basilar membrane as the resonating membrane. Second, was the conduction of sounds by the middle ear and the absence of sounds when this conducting mechanism was eliminated.

Dr. Sonnenschein asked if it would be feasible to convey sound by bone conduction alone by the fixation of an apparatus of some sort to the head of an animal after destroying the middle ear. It was assumed by Bezold that the only difference between air and bone conduction is that in air conduction the waves strike the flat surface of the tympanic membrane, whereas in bone conduction the sound waves impinge upon the edge thereof.

Dr. J. HOLINGER (closing the discussion) said that experiments had been made with two different whistles, one of lower and one of higher pitch, at the same time. In those experiments two areas of degeneration had been found separated by a normal part of the organ of Corti.

The fixation of the labyrinth was carried out by injecting the fixing fluid into the vascular system of the living animal, thus killing the animal. This was the quickest and best fixation that could be obtained and explained why the specimens were so complete.

The controversy between Siebenmann and Yoshi on one and Wittmaack on the other side had lasted for many years. Wittmaack attributed the changes in the cochlea to bone conduction. He argued that the animal was sitting on a sheet of metal, and the sound went through the bones of the body to the labyrinth. As a matter of fact, he exhausted the animals by continuously shaking them, day and night, plus the excessive sound. Siebenmann exposed his animals only eight or twelve hours a day, a period similar to the working hours of boilermakers and engineers. To prove that air conduction and not bone conduction produced the changes, the animals were placed on cotton or some other sound absorbing substance, so that no sound could pass from the cage to the animals' bones. In this way, by air conduction exclusively, he produced the same changes.

This is of great importance for the prevention of locomotive engineers' and boilermakers' deafness: If the floor upon which these men stand was responsible for the transmission of sound and therefore for the deafness all that would be necessary would be to put a nonsound conducting material on the floor for the man to stand on. This was tried and proved of no avail. Wittmaack had furthermore suggested that soldiers during artillery fire should either stand on their tiptoes, or on soft soil, but this too had no effect.

It soon became evident that the Japanese with their nimble fingers were the proper technicians for this work. The handling of the extremely delicate specimens, especially of birds, was very difficult. But for the mapping out and planning of the work, for criticising the experiments, passing judgment on the specimens and the composition of the text, Siebenmann's thought, work and even style can easily be recognized everywhere in the works published under the names of Yoshi and Satah.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, February 5, 1923.

THE PRESIDENT, DR. C. H. LONG, IN THE CHAIR.

DR. SAMUEL SALINGER presented a boy afflicted with a

Vitreous Abscess Following Nasal Sinus Thrombosis.

The case was presented because it was an unusual complication. The sinus phlebitis developed about two weeks after the onset of a suppurative otitis media with fairly typical symptoms, except that the chills were absent. The day before operation he began to develop pains in the ankles and other joints. At operation they found a mastoid abscess, a large extradural abscess and a lateral sinus thrombosis with pus within the lumen of the vein. The abscess was cleaned out and the internal jugular ligated. (Cultures from the mastoid as well as the thrombus showed chiefly staphylococci.) Four days later the boy developed a swelling of the right lids, both becoming very edematous and the conjunctiva greatly injected. The humors were clouded so that the fundus could not be seen. It was thought that the boy was developing a cavernous sinus thrombosis, but because there were no symptoms of involvement of the extraocular muscles and in the absence of exophthalmos it seemed that this might be an error. In a few days the swelling diminished, the anterior chamber cleared up and one could distinguish a vitreous abscess. At the time of presentation the eyeball was somewhat shrunk and the yellow accumulation within could be clearly seen through the cornea and unimpaired lens.

Dr. Salinger had never met with a vitreous abscess following a lateral sinus thrombosis. The boy has practically no light perception, and the Doctor has been informed that the sight is permanently lost, but there is no metastatic danger from this source.

DISCUSSION.

DR. HARRY WOODRUFF, Joliet, said that he never had seen an abscess of the vitreous following lateral sinus thrombosis but saw no reason why it could not occur. In any situation where pus might be present in the circulation such an abscess might form. He thought the patient was fortunate not to develop an abscess in both eyes, as occasionally happens following certain infectious diseases, such as scarlet fever. He was surprised that such a case had not been reported before. The fact that the pus cells in the circulation more often locate in the joints and do not affect the eyes he thought showed that they were well protected against this invasion. In his opinion, if the eye atrophied it should be removed.

DR. BURTON HASELTINE was much interested in the case because he saw an apparently identical one ten or twelve years ago. In that case there was a lateral sinus thrombosis, and operation was advised. The patient was seen the following day by Dr. Ballenger, who advised the same operation, although he did not know that Dr. Haseltine had previously seen the case. They both believed that the patient would die, as they thought it was a cavernous thrombosis. The patient did not die, however, and Dr. Haseltine saw her five years later, at which time Dr. Wilder also saw the patient. There had been no operation of any kind, but the patient had been ill for several months and had had numerous metastatic abscesses, some in the buttocks and various places throughout the body. When seen five years later, the eye was completely atrophied, but the patient had made a partial recovery from the arthritic condition.

DR. SALINGER (closing) said that this boy had two other metastases, one on the rim of the left auricle and the other on the scalp in the left occipital region. The operation disclosed an enormous amount of pathology, an extradural abscess, phlebitis extending from the knee to the bulb and a suppurating thrombus within the sinus. He was urged to operate again when the eye condition appeared, but felt he was fortunate in not having followed that advice. The boy was on the eve of recovery and was about to be dismissed from the hospital.

Paper: "Lymphoid Tissue of the Tonsillar Fossæ Following Tonsillectomy."*

BY SAMUEL J. PEARLMAN, M. D., AND
BENJAMIN J. GRICHTER, M. D.

DISCUSSION.

DR. JOSEPH C. BECK said he had so many times seen tissue left by the beginner in the use of the ring for the removal of the tonsil that he was skeptical about the development of follicular tissue from the picture of Tilley's work or the work of Poynter in Nebraska showing that there may be recurrences from the plica, as he showed the large lymphoid tissue in the plica which is seen after thorough tonsillectomy. Dr. Beck thought one should be careful about diagnosing a recurrence of tissue after a thorough tonsillectomy. If the essayists had stated that after operation the cavity was thoroughly inspected and those follicles which Tilley's picture showed were there microscopically it might be taken seriously, but if such a well worked up thesis were allowed to go by without some dissension it would give rise to claims of curing tonsil stumps with X-ray therapy. He thought some of those present would recall that at a very large gathering of the American Radiological Society work on tonsil stumps done by Dr. French and others in the East was shown to prove that operations for the removal of tonsils were useless because they recur. Some years ago Dr. Goode read a paper before the society showing that following tonsillectomy there was retro-tonsillar tissue. In Dr. Beck's opinion, many of these follicles were the remains of tonsils, whether the operation was done by ring, dissection or any other method.

DR. ROBERT SONNENSCHNIG thought a great many would agree that in many cases of complete tonsillectomy, so far as the eye could see, where the tonsil fossa had been inspected by pulling back the anterior pillar and where the fossa seemed as clean and as smooth as possible, weeks or months later one could see small round glistening masses similar to those seen on the posterior wall of the pharynx. In some such cases as those described by the essayists he had seen the fossa immediately after operation, with the pillars retracted, and it was

*See page 860.

as smooth as could be, so to the naked eye there was no sign of anything left. Then with a punch forceps these pieces of tissue were removed and examined microscopically and the conditions described were found.

DR. L. W. DEAN, Iowa City, Iowa, said that he felt that when tonsils were removed because they were known to be a focus of infection, the plica triangularis should be completely removed with the tonsil. In children with diseased tonsils without apparent systemic infection, an attempt should be made to save the plica and get a better cosmetic and functional result so far as the throat is concerned.

DR. CHARLES ROBERTSON recalled the case of a little boy, aged 12 years, who came into his clinic with immense tonsils and adenoids. This boy was of such a nature that he did not object to being operated upon and consequently was demonstrated before a class. He performed complete enucleation of the tonsils and complete ablation of the adenoids. In three weeks he returned and presented the appearance of an immense tonsil on either side, with a large adenoid in the nasopharynx which was fully as large as the original one. He was again operated upon and the masses removed as completely as they could be removed, so that one could see the fibers of the constrictor muscles. In about three weeks the patient returned with exactly the same condition. The masses were removed a third time, and following that removal there was no recurrence, but there was a supernumerary tonsil behind each pillar, a beginning tonsil just behind the uvula.

Dr. Robertson thought that in these cases it made little difference how thoroughly and completely the tonsils were removed, as some of the growths recur, and recur in different degrees in different patients. An incision of the skin of one patient will heal without a scar, whereas another patient will have a keloid scar. He had paid particular attention to this question and believed there was a systemic condition which was at fault and which should be investigated. In his opinion, there is a certain condition in which lymphoid tissue and granulation tissue piles up. In the mastoid in one case one will have to burn granulations, while in other cases there will be no granulation tissue to burn, while the wound will need stimulating. One case is exuberant and the other anemic.

He had seen in some cases an exuberant hypertrophy in two or three weeks, which eventually shrunk, leaving a nice smooth cavity.

Dr. Robertson believed that no one could say that the lymphoid tissue developed a crypt, or whether it was pure granulation tissue or a piling up of lymphoid tissue from one spot. He thought much work had to be done on the tonsillar fossa, and no one at present realized the seriousness of the proposition. He had noticed that in particular cases, for instance, in people who were suffering with tuberculosis, if they were operated upon there would be an exuberant granulation tissue pushing up into the tonsillar fossa or some place else. He had seen many such cases. No one had ever studied the matter to determine whether there was anything in the blood that had to do with the piling up of this tissue, and Dr. Robertson thought this offered a field of investigation that would be productive of good results.

DR. EDWIN MCGINNIS thought this was not a postoperative recurrence of the tonsillar tissue but, as he understood it, pieces of lymphatic tissue which were clipped out of the spaces behind the tonsils. He could confirm these findings in some work that he had done. He did not use the Beck ring, because he felt that it had a tendency to slip, and one might have a fringe of tonsillar tissue left around the wound. In the Sluder operation one often finds the upper part of the tonsil slipping out of the fenestrum and in enucleating the tonsil this is frequently left behind. Sluder has said that occasionally he would buttonhole a part of the tonsil, and it was a question whether there would not be a recurrence from that part of the capsule which was left.

Dr. McGinnis had had some cases which he had been watching to see whether they would develop a tonsillitis and whether the tonsils would regrow. He believed all would agree that in the last few years there had been fewer recurrences because better work was being done. On removing tonsils under local anesthesia, in dissecting back over the top one will frequently find a little lymphoid tissue that slips away from the capsule, and if these pieces are snipped off they prove to be lymphoid tissue. Dr. McGinnis operated on one patient two years ago, doing a very thorough operation, and this patient returned

about two weeks ago with hypertrophy of the lateral mass of the lingual tonsil and a tonsillitis in the hypertrophied part of the tissue.

DR. GEORGE L. RICHARDS, Fall River, Mass., agreed with Dr. Beck that with either the Sluder or any other type of operation it was easy to remove the tonsil and, after passing an instrument all around in the tonsillar fossa, to conclude that one had performed a perfect tonsillectomy. On the other hand, if with a dull retractor one elevated the anterior pillar so as to expose the whole supratonsillar space there would frequently be brought into view a small remnant of tonsillar tissue.

Some years ago, when first doing tonsillectomies, he made a good many microscopic sections and found that the muscle tissue extended into the capsule and that the gross appearance of this tissue on the capsule was by no means an indication, as claimed by some men, of too extensive removal of the tonsil.

Thorough inspection of the entire tonsillar fossa by retraction of the pillars was the only safeguard against leaving behind unrecognized portions of tonsillar tissue.

DR. JOSEPH D. HEITGER, Louisville, Ky., said that in 1914 Grunwald published an article in the *Archiv. für Laryngologie und Rhin.*, dealing with the tissue Dr. Richards spoke of and apparently in the location from which the essayists said they had removed the tissue. Grunwald described five main types of tonsillar formation dependent upon the relation of the plica transversa to the upper and lower poles of the tonsil. The various plicæ undergo various types of change, which influence very markedly the development of the tonsil. Grunwald attributed the aberrant portion of the tonsil tissue, simply the portion snared off, to the development of these various plicæ, but the percentage of these variations was very much lower than that found by the essayists.

DR. ELMER L. KENYON called attention to Dr. Richards' remark concerning muscle tissue being found in the capsule and said it was perhaps not commonly known that lymphatic tissue penetrates and forms islets in the capsule. He wondered whether it might not be found upon careful microscopic study that these islets completely penetrated the capsule, and

thought this might explain why lymphatic tissue was found outside the capsule.

DR. SAMUEL J. PEARLMAN (closing) agreed that there was such a fold of tissue as Dr. Richards had described, and it might be that in some instances some of it had been taken for study. However, they inspected the tonsil and fauces as carefully as possible, and many cases did not present these findings. They took sections along the course of the anterior plica, not always up in the angle but always at least half way up, so that there was never any question of getting tissue around the lingual tonsil. He believed there was sufficient anatomic reason for finding this tissue, even after apparently complete removal. Some of the tissue was microscopic and could not be seen with the naked eye. Occasionally they found very small follicles under the microscope; other tissue was diffuse in character. They stated in the paper that they did not know whether this lymphoid tissue would atrophy or hypertrophy and also stated that anatomically lymphoid tissue is often present following apparently complete removal of the tonsils. There is lymphoid tissue in this region continuous with the tonsil and performing the same function, but in all instances it cannot be removed with the tonsil. He did not think the tissue they examined was granulation tissue, as it had none of the appearance of granulation tissue. They had done the best they could, but anatomically it was impossible to remove tonsils 100 per cent by any method whatever.

Paper: "Differential Diagnosis of Labyrinthitis Syphilitica Tarda and Neurolabyrinthitis Syphilitica."*

By GEO. W. MACKENZIE, M. D.,

PHILADELPHIA.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH said that he expressed the sense of the society in saying they were all very grateful to Dr. Mackenzie for coming to Chicago and that they all appreciated very much his clear, lucid discussion of an interesting condition. His presentation was so clear that no one could have difficulty in following him.

*See page 847.

Dr. Shambaugh asked Dr. Mackenzie what he had to say about the use of arsphenamin in luetic cases. Some observations have been made which suggest that this drug may have a very specific effect upon the eighth nerve. He could recall three cases where a patient afflicted with lues had developed labyrinthine symptoms with a more or less profound destruction of the hearing and where the administration of arsphenamin had been followed rather abruptly by a very marked exacerbation of the labyrinthine symptoms. The idea that an improvement follows further use of the drug had not shown itself in these cases.

DR. J. GORDON WILSON said he had listened with great pleasure to the interesting and instructive paper of Dr. Mackenzie. He thought there was no doubt that theoretically electrical stimulation is better than either caloric or rotation stimulation. His experience was that in man the results of electrical stimulation were divergent and not sufficiently sharp and reliable. He used three to four milliamperes and so avoided discomfort and pain. Within the last month he had occasion to observe in man in erect posture electrical stimulation of the vestibular mechanism with such a current and noted that while typical falling reaction was prominent and some vertigo was present there was very little or no nystagmus.

In regard to the hereditary cases, it is recognized that the majority of them become apparent between the eleventh and seventeenth year, usually associated with syphilitic stigmata in other parts of the body. Those are the cases which present the anomalies in regard to hearing and pressure symptoms. In the case of eighth nerve lesions in acquired cases of syphilis, one meets with three varieties: First, where both functions are affected; second, those in which disease falls chiefly on the acoustic apparatus; and third, those which involve chiefly the vestibular. The latter are frequently delayed in their onset, coming first into notice with a crisis.

In regard to treatment, Dr. Wilson had seen, as many others have, acute exacerbations after the use of hypodermic doses of salvarsan, so he now prefers to use mercurial treatment before using salvarsan. In hereditary cases treatment is unsatisfactory and in marked contrast to the results obtained in acquired cases.

DR. ALFRED LEWY said that some years ago he undertook to follow up some of Dr. Mackenzie's investigations of the auditory nerve with the galvanic current. He thought he had learned the proper technic, but found that the attempt became so painful to the patient before a diagnostic conclusion could be arrived at that the test had to be discontinued. He had given up the attempt because the patients did not tolerate it. He thought the tests probably necessitated a special type of apparatus in which the current strength could be absolutely controlled, certainly better than the usual rheostats do it, and a milliamperemeter of greater accuracy and delicacy than the ordinary. The instrument which he used was attached to the street current, and this constituted another difficulty, as the current, at least in Chicago, varies suddenly according to the demands on it. His apparatus was not capable of controlling it to the fine degree of precision necessary to make this delicate test.

Dr. Lewy asked Dr. Mackenzie, in closing, to describe in detail the type of apparatus he used and to state whether or not he used the street current or some type of battery that could be controlled more accurately.

DR. L. W. DEAN, Iowa City, Iowa, said he had been interested in the galvanic tests of Dr. Mackenzie for many years. He asked Dr. Mackenzie if the late manifestations of syphilis occurring in tabes were shown by his tests to be labyrinthitis or neurolabyrinthitis.

DR. JOSEPH D. HEITGER, Louisville, Ky., said that his experience was similar to that of Dr. Wilson. He had talked with Dr. Mackenzie and read his book, which he published in 1910, and had tried out the street current, without results. Finally he went down to Dr. Mackenzie and took up the work with him. Dr. Heitger had been looking for a large nystagmus, such as would be obtained in the use of the caloric or turning test, but it was pointed out that the real nystagmus to look for was just a tiny little jerk. He accordingly had an apparatus constructed under the direction of the electrician and Dr. Mackenzie which had a very delicate milliamperemeter, rheostat and polarity changer, and with this a good deal of the pain mentioned by Dr. Lewy could be eliminated by getting the reaction at just the moment it should be obtained, having

the electrodes, the patient's hands and tragus well soaked with salt solution. The greater resistance that is offered, the more pronounced the pain.

A point which had occurred to Dr. Heitger while listening to the paper was that Dr. R. Foster Moore, in a recent book, made the statement that interstitial keratitis could not be prevented by any form of treatment. This was a rather broad statement and, coming from a man of Dr. Moore's experience in ophthalmology, it must be accepted. It was a question with Dr. Heitger whether any of the tarda forms could be prevented even if detected early. In a series of studies of congenital syphilis in the Department of Pediatrics in the Barnes Hospital of St. Louis, including several hundred cases, it is of considerable note that only three cases of the classical Hutchinson triad were found.

Another point was that probably many of the cases slipped through the examiner's hands because a complete functional examination was not always made. A patient might come in complaining of deafness in one ear only, but careful examination might reveal that both ears were affected, one more than the other. Dr. Heitger thought if both ears were tested thoroughly, both with the functional tests of the cochlea and the other tests as given by Dr. Mackenzie, all would have the same experience that he had had, namely, that it was a matter of learning the technic and controlling it carefully.

DR. NORVAL H. PIERCE said that Dr. Mackenzie exemplified the qualities of his stock which had caused it to persist so long in the bleakest portion of the world, namely, persistence. He had been advocating for years and years the use of the electric reaction in cases of labyrinthine trouble, but for some reason or other it had not become popular. Dr. Pierce was rather inclined to think that the Viennese school in the early days looked askance at it, and perhaps this had had some effect on its general acceptance. He had had the experience that Dr. Lewy and others probably had had. The pain and uncertainty had been caused by an improper apparatus, but that the test was of great value he was convinced and believed the profession was to blame for not using this valuable means of diagnosis more frequently. Dr. Pierce thought Dr. Mackenzie's valuable paper would bear fruit and that the tests would

be used more frequently, especially if one could differentiate diseases of the terminal apparatus and of the nerve itself. He did not believe that the profession was absolutely dependent upon this method for this differential diagnosis, for the very therapeutic tests Dr. Mackenzie recommended would locate the syphilitic disease. If under mercury the patient recovered there was no doubt that the disease was meningitis; if not, the disease was in the labyrinth.

Dr. Pierce asked Dr. Mackenzie to tell the society what was the best kind of apparatus and the best method by which this means of diagnosis was carried out.

DR. SAMUEL SALINGER, in connection with the discussion as to the apparatus, asked if Dr. Mackenzie had ever tried applying the galvanic current direct to the drum membrane. Dr. Salinger had done this by means of a sponge placed directly in contact with the drum membrane and had found it possible to obtain a reaction with a current of not more than one or two milliamperes. He was convinced that if one had the proper milliamperemeter they could obtain the reaction with less pain than was possible by the other method and without having to pass the current through the enormous amount of tissue that is necessary when the electrode is applied to the tragus. He was interested to know whether Dr. Mackenzie had ever attempted the test in this way.

DR. JOSEPH C. BECK referred to the treatment in the hereditary tarda cases. In the past year he had observed four cases which were very interesting. One was a meningeal case late in life, in which there was a bilateral deafness, both labyrinths being knocked out, with bilateral facial paralysis. He assumed that this condition extended through the internal auditory meatus. This sort of case was of great importance from the point Dr. Shambaugh had mentioned. The patient was placed on arsphenamin, and Dr. Beck had never seen a patient who responded more readily to the intravenous use of this drug. The condition cleared up and the patient left the city, but three weeks later he received a letter from him stating that he was completely deaf and that the face was immobile again. Dr. Beck thought Dr. Mackenzie would recall the work of Dr. Benario showing that swelling of the nerves in the narrow canal occurred in the early part of the treatment, but if fol-

lowed by more arsphenamin this condition would clear up. This had been the experience of many men, among them Hata, who claims to be the man who discovered salvarsan, and Dr. Beck thought this an important thing to bring out. He had had several cases of hereditary tarda in which the symptoms mentioned by Mackenzie were present. He had tried the galvanic current and the man screamed with pain as soon as the current was turned on, claiming that he felt severe pain at the site of application of the current. Dr. Beck was interested to know what type of apparatus Dr. Mackenzie had used that the patients could stand as much as 20 milliamperes.

In a case of hereditary tarda with marked keratitis they placed the patient on a chair and surrounded her with a rubber cover and burned calomel, allowing the calomel fumes to cover the patient's body. It seemed that this form of mercurialization had a very desirable effect on these deaf patients. Dr. Beck had recently seen a boy of about 20, who was absolutely deaf, who gave no reaction to cold water or turning and who had a severe labyrinthitis. Three months later he had a negative Wassermann reaction. That man's ear improved markedly on some days and then again he would be practically stone deaf. He had no reaction to turning, and when the electric current was applied he complained of severe pain and did not tolerate the test.

Dr. Beck expressed his gratitude to Dr. Mackenzie for his excellent paper.

DR. ROBERT SONNENSCHN said that the change in the bone conduction in early cases of syphilis had been noted, especially by Oscar Beck and others. It was characteristic that before the hearing changed to any degree the bone conduction was very markedly changed. Dr. Sonnenschein asked Dr. Mackenzie whether in the cases where the ear was markedly impaired the change in bone conduction threw any light on the differentiation between labyrinthitis and neurolabyrinthitis.

DR. HARRY WOODRUFF, Joliet, Ill., speaking in regard to involvement of the eyes, said he had been rather struck with the infrequency with which one encountered involvement of the labyrinth with interstitial keratitis. Interstitial keratitis from hereditary syphilis was one of the more common eye diseases—in fact, phlyctenular keratitis and interstitial kera-

titis form the most common diseases of the cornea in childhood. It had been mentioned that one seldom finds the Hutchinson triad in these cases and that there was lack of results from any form of treatment, and Dr. Woodruff thought the ophthalmologists would agree with this statement. Some had gone so far as to advocate not treating these cases at all, but he always treated them energetically with some form of mercury. It was true that during the treatment of these cases the second eye would nearly always become involved, notwithstanding the thoroughness of the treatment. The results in these cases were not so bad, so far as the cornea was concerned. It was seldom that permanent and complete loss of vision occurred. Dr. Woodruff recalled a case which he had had under observation for many years with interstitial keratitis and also involvement of the labyrinth. The cornea cleared up very well and the patient had useful vision, while the labyrinth never recovered, although it had not reached a stage that incapacitated her. She had thorough treatment over a long period with apparently no effect on the labyrinth but a good effect on the cornea. Whether this was simply the result of time or of the mercury he could not say.

DR. THOMAS FAITH agreed with Dr. Woodruff regarding the appearance of keratitis in the second eye. He recalled that some years ago it was a common notion not to treat syphilis in interstitial keratitis but to regard the disease as postsyphilitic and improve the general health in every way possible, clearing up the indications of the disease as far as possible.

DR. GEO. W. MACKENZIE (closing) expressed his pleasure with the discussion and thanked the gentlemen who took part in it.

Regarding Dr. Shambaugh's question about exacerbations following the use of arsenical preparations, Dr. Mackenzie recalled that just after the International Congress in Boston where Dr. Beck took part in the discussion of the vaccine treatment of middle ear suppuration, the question arose as to what arsphenamin might do to the optic nerve. At that time Dr. Beck published a paper in which he stated his experience with the Herxheimer reaction and other things, including neurorecurrences. He thought this would answer Dr. Shambaugh's question. Before the day of arsphenamin many other

preparations were used, and the big objection was that the arsenic hit the central nervous system too often and too hard, more particularly the cranial nerves, so that Hata, with Ehrlich, tried to obtain a synthetic arsenic preparation that would not do these damages. Their success was challenged for a while when some odd cases of damage to the second and eighth nerves were reported. But in those cases it was found that upon pushing the arsenic preparations still further and harder, these conditions that appeared to be caused by the arsenic cleared up, showing that the apparently bad results were produced by insufficiency of treatment rather than by the salvarsan itself.

Regarding Dr. Wilson's question about the 20 milliamperes, Dr. Mackenzie said that in the paper he stated that he used 20, or as much as the patient could tolerate. Very few of them could tolerate more than 10 or 12 ma. In a deafmute institution in which he did some work he pushed the milliamperage up to 20 in those cases where the patient was particularly anxious to cooperate, but for practical purposes this strength was not necessary. He thought one did not need to use more than 8, but occasionally he goes as high as 12. He had tried 20 on himself and admitted that it was very painful. Dr. Wilson said that his patients did not complain so much of the pain as of the falling reaction. If the technic was good Dr. Mackenzie was sure that it was possible to get reaction that could be observed with the eye long before the appearance of vertigo, and any patient who had a falling reaction because of the galvanic current already had marked nystagmus, the difficulty being that the operator did not recognize it. No one can look into a patient's eye at a distance and tell through the ordinary palpebral fissure just when the nystagmus begins. The nystagmus obtained with the galvanic test is a mixed rotary and horizontal nystagmus, not one form alone but mixed, because the end organs are being stimulated in all three canals.

The first requisite in making this test is a good light. The operator must get into a favorable location, with a head mirror in position and, standing in front of the patient, throw the light into the patient's eyes obliquely. Next, the head must be tilted very slightly backward with the lids elevated by

the thumb of the examiner, so that the equator of the globe may be observed. A movement of $\frac{1}{2}$ mm. at the corneal limbus is equivalent to 2 mm. at the equator. With the head tilted backward and lid elevated, one can distinguish well what would be a very tiny nystagmus at the corneal limbus. The question has been asked, why not try for a big movement? The same objection applies here as in the case of after-nystagmus. In after-turning nystagmus one will notice a wide excursion nystagmus immediately upon stopping turning, which can be seen at a considerable distance through the normal palpebral fissure, which gradually slows down until it ceases altogether. The most essential thing to do is to time the nystagmus, and if one is not a keen observer he will surely miss the fine movements toward the end of the reaction or, say, after fifteen seconds, when he is apt to record the after-nystagmus as fifteen seconds. The careful examiner should study the movements in the after-turning test until they cease altogether. From the fifteenth second on to the twenty-fourth, the movements will be observed to become finer and finer until it becomes a question whether there is any movement or not. He thought it was well to use a split second stop watch and snap the moment the examiner thinks the movements cease and again when he is dead sure they have. It is just as difficult to time the after-nystagmus as it is to make the galvanic test. If one produces with galvanism a big, coarse nystagmus, the question of the personal equation enters too much. For instance, the observer is left to judge the length and frequency of the excursion when they are equal on the two sides. That is surely poor technic. The proper thing to do is to use the least amount of milliamperage that is required to produce the least amount of nystagmus. Nystagmus which is just as fine as that noted after the sixteenth or seventeenth second in the case of after-turning nystagmus. Otherwise the test would be of no value. The same thing applies to the after-turning nystagmus in that we time the least amount of nystagmus.

Dr. Mackenzie thought he might have left some confusion in the minds of some of those present as to the types of syphilis of the temporal bone. He had described during the last several years the types of syphilis he was best acquainted with

and which were possible of differentiation by the galvanic test. There are other forms which he did not feel prepared to discuss at this time. In Providence a week previously Dr. Mackenzie spoke of some other forms that he hoped to put on a sounder basis than they are at present. Parasyphilis or metasymphilis is not tardy syphilis; they are entirely different conditions. Neurolabyrinthitis is not syphilitica tarda, but the result of syphilis of the meninges that every syphilitic patient manifests more or less during the secondary stage. Syphilis is pathologically a perivascular round cell infiltration. This character of infiltration is found more or less evident in all granulomatous inflammations but not so characteristic as in syphilis. When a patient has syphilitic meningitis of the arachnoid, and more especially of the pia, there is not only the meningitis present but meningoencephalitis. Later there is cicatrization, and nothing scars like syphilis. After several years of contraction of the previously inflamed membrane and cortex, atrophy of the cortex of the brain results; in other words, paresis. The same explanation holds in the case of the spinal cord. In the Lissauer zone, where there is no medullary sheath to the fibers of the posterior roots, we have a place where the resistance is weakest that is most affected, and when the pia shrinks it gives rise to the so-called lightning pains of tabes. If it happens to hit the fifth nerve the patient is liable to have (in the cervical type of tabes) tic douloureux. Dr. Mackenzie recalled a case in which the Gasserian ganglion had been operated on and removed. The patient subsequently was seen in the Neurologic Clinic and, in spite of the fact that the Gasserian ganglion had been removed, the patient still had pains because the Gasserian ganglion is peripheralward of the roots of the sensory part of the fifth nerve. The same process takes place in the eighth nerve, when we have a primary atrophy, due to the effects of the original neurosyphilis and the subsequent contraction that takes place in the meninges. Retrolabyrinthine primary atrophy of the eighth nerve is comparable with primary atrophy of the optic nerve. Dr. Mackenzie did not care to go into this fully at present, because he is still working on it and will not be prepared to give the most definite findings for some years. This is perhaps the condition that Dr. Dean had in mind. In these cases the deafness is due

to primary atrophy of the nerve and not to the Hutchinson's syphilitic triad. Primary atrophy is due to the late effects of earlier neurosyphilis.

Regarding Dr. Heitger's statement concerning the fact that they found only three cases of the Hutchinson triad in 700 cases of congenital syphilis studied in St. Louis, Dr. Mackenzie said that the reason for this was that if a child was affected at 13 years of age with a syphilitic keratitis, that child was usually sent to the eye clinic. If it happened to be affected with difficulty of hearing it was sent to the ear clinic. For this reason very few cases of Hutchinson's triad were found in the syphilitic clinic because the parents probably knew little of the original cause of the eye or ear trouble. Dr. Mackenzie thanked Dr. Heitger for making clear his early experiences, and then his later experience with the galvanic test. He considered Dr. Heitger a real scientist who, when he knew a person had something worth while, made a trip to that person's clinic and stayed there until he had learned all he could of the other fellow's technic. He thought Dr. Heitger would bear him out that he had found the nystagmus present before the patient complained of vertigo and without any falling reaction. If the patient was asked if the test was painful, he would answer "no." Some children are a little fearful if one starts with electricity, but after obtaining their confidence the test can be made quite readily. He invariably makes the galvanic test, and where it is necessary makes all three, and then asks the patient which was the most painful. They invariably say the caloric test is the most distressing, no doubt because it produces, with the head erect, rotary nystagmus; besides the caloric test is more unpleasant than the galvanic test, for the reason that it is less under control and it is prone to produce vomiting and severe vertigo that is hardly possible with the galvanic. He believed the least unpleasant test of all was the galvanic.

As to the technic, the observation of the nystagmus was of the very greatest importance. Dr. Mackenzie uses an electric apparatus made by Geiger of Philadelphia, which was designed for his particular purpose. He formerly used the current from the Professional Building, in which his office was located, which generated its own current and which was sup-

posed to be 110 volts but really averaged about 90 volts. He has an ammeter which is full jeweled, the same as a full jeweled watch. As to voltage and milliamperage he did not know the exact difference, whether 220 or 110 volts would produce any difference in effect so long as the milliamperage remained the same. Where he is located at present he uses the street current of 110 volts. So far as the ammeter readings were concerned, he could see no difference in the reaction between the current formerly used which tested out at 90 volts and that which he is using at present, which is 110 volts. He was not expert enough to answer the questions raised by Dr. Richards. In his opinion the apparatus could be used with the street current because he is now getting just as satisfactory results as when he used the 90 volts.

A very essential point in the work is to see that the electrodes are perfectly wet. He uses normal saline solution; why, he does not know, except that he had been told by electrical experts and neurologic experts that they used salt solution, and he had adopted it empirically. He has the electrodes dripping wet, with a small ball electrode for the ear with contact that can be made and broken at will. He has a polarity switch in connection with the instrument. It is necessary to have an assistant to control the current and read the meter. He does not know the polarity while making the test, but the moment he observes the nystagmus he notes the polarity. He starts with a low current and is never satisfied with one test. If a questionable reaction is observed at 3 or $3\frac{1}{2}$ m. a. and at 4 or 5, the movements are unmistakable, we know the reaction is positive somewhere between $3\frac{1}{2}$ and $4\frac{1}{2}$ m. a., when we generally credit it as 4. He is not satisfied with one test alone, but repeats the test. It is absolutely necessary to have a good light, to look for eye movements at the equator of the globe, to use a very small ball electrode, or put the electrode into the canal, ignoring the tragus, and to have the electrodes very wet.

As to the electrode applied to the tympanic membrane, Dr. Mackenzie saw no objection to this and hoped that Dr. Salingler would continue using it in that way and give the results of his findings at a later date.

Dr. Mackenzie had obtained some very definite results with 4 m. a. of current. It is known that if a human being is hit in the head with a brick or shovel or the skull is fractured from any cause whereby the petrous bone is not only broken but the fragments are displaced and overriding and the patient bleeds from the ear, not only blood but spinal fluid, then the inner ear must be out of function. In cases of this kind we find mixed horizontal and rotary nystagmus, to the opposite side, with complete deafness. Why does such a patient have nystagmus to the other side? The patient has mixed horizontal and rotary nystagmus to the other side because he has lost the tonus from the ampulla of all three semicircular canals, which was present when the ear was intact. The tonus is lost immediately the labyrinth is destroyed and the balance is in favor of the other side. The amount of loss can be measured with galvanism. The minimum amount of alodal current to produce nystagmus to the opposite side comparable with destruction is 4 m. a. This indicates that the tonus loss by the destruction equals 4 m. a. He has verified this, for although the patient's labyrinth is destroyed the nerve stem is intact and reactive, and by applying 4 m. a. of cathode stimulation to the destroyed side the nystagmus to the opposite side is arrested. Applying 4 more m. a. to the destroyed side, making a total of 8 m. a. stimulation, produces a nystagmus toward the side of destruction.

Dr. Mackenzie extended a cordial invitation to anyone who happened to be in Philadelphia to visit him and said he would be glad to go over the galvanic test with them and demonstrate the technic.

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LXX.

CONTRIBUTION TO THE PATHOLOGY OF THE LABYRINTH.*

By J. S. FRASER, M. B., CH. B., F. R. C. S. ED.,

SURGEON, EAR AND THROAT DEPARTMENT, ROYAL INFIRMARY,

EDINBURGH.

MR. PRESIDENT AND GENTLEMEN :

Before entering on the subject of labyrinthine pathology, I should like to say a few words about the method by which the inner ear may be microscopically examined. Although this method is a tedious and often disappointing one, I hope that some of the younger otologists in this country may be induced to take it up. There are still large gaps in our knowledge regarding the pathology of the labyrinth. For instance, no one has yet, as far I know, described the microscopic pathology of deafness due to mumps. Neuritis of the eighth nerve following infectious fevers has not been fully elucidated. The pathology of old age deafness is still in dispute—some observers ascribing it to degenerative changes in the cochlear gang-

*A paper read before the American Laryngological, Rhinological and Otological Society, Atlantic City, May 11, 1923.

lion cells and in Corti's organ, others to calcification of the basilar membrane. The changes in the inner ear in congenital and acquired syphilis have not been fully explained on a pathologic basis, nor has the neuritis which is so commonly observed in cases of toxemia from various causes. I could add to this list, but I have said enough to demonstrate the need for further investigation. I earnestly hope that American otologists may soon be able to fill in the gaps in our knowledge.

1. THE MICROSCOPIC EXAMINATION OF THE MIDDLE AND INNER EAR BY THE CELLOIDIN METHOD.

Preparation of the Block of Bone Containing the Middle and Inner Ear.—The temporal bone must be obtained as soon as possible after death—certainly within twenty-four hours. In removing the brain the seventh and eighth nerves should be cut close to the pons, so as to leave as much as possible attached to the temporal bone. The dura mater is cut with a knife round the internal auditory meatus in order that the nerves may not be pulled out when the dura is stripped off. The sacculus endolymphaticus is examined with the naked eye, and for this purpose a crucial incision is made into it. The dura mater is now removed with forceps from the upper and posterior surfaces of the petrous bone.

With a small butcher's saw a cuboid block containing the important parts of the middle and inner ear is cut from the temporal bone. The first saw cut is made in a vertical direction at right angles to the long axis of the petrous pyramid just in front of the internal auditory meatus. The second cut is made parallel to and behind the first, through the mastoid antrum, posterior to the semicircular canals. The third vertical saw cut is made in an anteroposterior direction through the middle cranial fossa and external auditory meatus and runs parallel to the posterior surface of the petrous pyramid and to the middle ear cleft. The last cut runs through the jugular fossa and is made in a horizontal direction below the middle and inner ear.

In this way a six sided block is obtained, but it requires some trimming. The anterior wall of the external meatus should be cut away with bone forceps, so that the tympanic

membrane may be exposed and its condition noted. The jugular bulb is also inspected and thereafter its lining membrane is removed with dissecting forceps. The carotid canal is opened up with bone forceps and the artery removed—care being taken not to injure the eustachian tube, which lies just external to this vessel. Finally, the superior semicircular canal is opened with bone forceps in order to allow the fixing fluid to gain access to the inner ear. (It must be noted that the superior canal lies somewhat in front of the arcuate eminence.) The block so obtained is washed in running water to remove bone dust and is then placed in a wide mouthed glass stoppered bottle containing 5 per cent formol or Jore's fluid for one month—the fluid being changed on several occasions. If the nerve structures are to be specially examined, it is best to place the block in Müller's fluid after it has been twenty-four hours in formol. The Müller's fluid must be changed frequently.

Decalcification, Washing and Hardening.—After the block has been fixed it may be decalcified in 5 per cent formol and 5 per cent acid (equal parts) for one month or six weeks, according to the size of the block. (Perenny's solution may be used instead—10 per cent nitric acid, 364 c. c.; methylated spirit, 336 c. c.; chromic acid, $\frac{1}{2}$ per cent, 300 c. c.) During the first week the fluid is changed daily, during the second week every second day, and during the third and following weeks the fluid should be changed twice weekly. After decalcification the block is gently removed and placed in a porcelain dish and washed in running water for four or five days. The specimen is next hardened for the second time, first in 70 per cent spirit for twenty-four hours, then 80 per cent for twenty-four hours, then methylated spirit for three days (this latter solution being changed daily). From methylated spirit the block is passed into absolute alcohol for two days (changed daily), and then into absolute alcohol and ether (equal parts) for two days (changed daily). During the hardening process it is convenient to use a series of ten or twelve wide mouthed glass stoppered bottles.

Embedding.—The block is now put into thin celloidin for one month—a bottle similar to the above being used—and then transferred to thick celloidin for one month. (During the

time the block is in the thin celloidin it may be an advantage to employ an evacuation pump so as to get rid of air bubbles from the hollow spaces of the middle and inner ear, but the negative pressure employed must not be excessive, otherwise the structures of the membranous labyrinth may be distorted. It is better to reserve the evacuation pump for cases in which one finds on microscopic examination that the hollow spaces of the ear contain air cells. The specimen should then be put back in absolute alcohol and ether for six hours and then transferred to thin celloidin before the pump is employed.) At the end of the second month of embedding, the lid of the glass jar containing the block is left partially open so that the specimen may gradually dry and harden. When the celloidin is thoroughly hardened—usually in about a week—the block is cut out with a knife and pared down with a razor to the required size—i. e., a cube measuring about three-quarters of an inch in all dimensions.

Cutting.—The block may be cut in one of two directions: (1) Horizontally, from above downwards; in this way some 400 sections are obtained, 20 micromillimeters in thickness. (2) The specimen may be cut vertically, from before backwards; in this way about 700 sections are obtained. After deciding in which direction the block is to be cut, it must be fixed on a piece of stabilit; the block is dipped into absolute alcohol and ether for a moment and then into thick celloidin and placed on the stabilit with the surface to be cut uppermost. The specimen should now be left for half an hour or more in the air so that the celloidin may harden and then placed in a bottle of methylated spirit for six hours. The stabilit block with the specimen attached is then removed and fixed in the Schantze microtome. If the block is very hard sections may sometimes be obtained 10 mm. in thickness, but as a rule 20 mm. give sufficiently thin sections. The writer has been in the habit of numbering and staining every fifth section only; the intervening ones are placed in a large fruit jar containing spirit. In very important cases, however, such as deafmutism or otosclerosis, every section should be numbered and stained. In less important cases the sections which are selected for staining are placed on a slide and wrapped up in a strip of blotting paper moistened with spirit; they must on no account

be allowed to dry. Before the blotting paper is put on each slide is numbered with a diamond, thus—"William Smith, 5, 10, 15, etc." As each hundred sections are cut, the selected twenty are tied up together in a bundle, labeled and kept moist with spirit. When all the sections have been cut they are placed in a large glass jar containing spirit. Staining may be deferred to a convenient occasion.

Staining.—The blotting paper is unwrapped and the section removed by tapping the slide against the side of a bowl containing water. The section is now lifted with a copper section lifter and placed in a watch glass containing filtered hemalum (Mayer's). In this the section remains for a period varying from five to thirty minutes, according to the quality of the staining solution. The section is then lifted out and thoroughly washed in a bowl of water, and then transferred to a watery solution of eosin ($\frac{1}{4}$ to $\frac{1}{2}$ per cent). The time taken for staining with eosin varies from two to ten seconds as a rule. The specimen is again washed in a second bowl of water and transferred to a third watch glass containing spirit, in order to remove the excess of eosin. From this it is passed on to a fourth and fifth watch glass containing spirit, and is then placed for a second or two in a sixth watch glass containing absolute alcohol. Finally, the specimen is removed to a seventh glass which holds carbol-xylol, where it remains for a minute or two until it sinks to the bottom of the glass. The section is then transferred with the aid of a needle and a copper section lifter to the numbered slide from which it was taken for staining purposes. It is now covered with Canada balsam, and with the aid of a needle a clean cover slip is slowly let down over it so as to exclude air bubbles.

Other staining methods may be employed—e. g., van Giesen, iron, alum, osmic acid, etc. For details of these methods the reader is referred to textbooks on histology.

It is possible to work three rows of watch glasses (twenty-one in all) containing the various solutions at one time, and in this way from sixteen to twenty sections can be stained in two hours.

It will be seen that the process required for the microscopic examination of the middle and inner ears is a somewhat lengthy one, taking as it does about six months. It must also

be confessed that many difficulties and disappointments are met with, notably injury to the ear in cutting out the block of bone, failure to decalcify sufficiently, presence of air cells in the hollow spaces of the middle and inner ear, and faulty staining. For all these reasons the microscopic examination of the ear is not a task to be lightly undertaken.

2. ANATOMY OF THE LABYRINTH.

It is not my intention to inflict on the society an account of the anatomy of the inner ear. It may be, however, that some of the members are not accustomed to examine microscopic sections of the labyrinth and consequently may find it difficult to recognize the various parts of the membranous labyrinth as they appear in sections. For this reason I propose to show a series of photomicrographs of the inner ear (of very low magnification) as it appears when cut (1) in vertical sections from before backwards, and (2) in horizontal sections from above downwards. (Figs. 1 to 16.)

3. INJURIES.—FRACTURE.

Fractures of the cranial base frequently involve the middle and inner ear. (There is disturbance of hearing in 24 per cent of the cases of basal fractures who recover.) The line of fracture may run parallel to the long axis of the petrous bone or at right angles to the long axis.

(1) Longitudinal fractures start in the region of the sella turcica, and pass backwards along the line of the middle ear cleft, breaking the roof of the eustachian tube and tympanic cavity. The fracture may then pass outwards to the external meatus and squamous region; if this is the case the inner ear is not involved, though the ossicles may be dislocated and the drumhead torn. On the other hand, the fracture after reaching the roof of the tympanic cavity may pass inwards through the petrous pyramid and thus resemble those fractures which run at right angles to the long axis of the petrous bone. The inner ear is of course involved in these latter cases.

(2) Transverse fractures of the petrous pyramid always injure the labyrinth. As a rule the fracture passes through the external meatus, roof of the tympanic cavity, vestibule and internal auditory meatus, as this is the line of least resistance. The fracture may, however, pass further forward

through the cochlea, or further back when the canals are involved.

If the patient survives the injury at the time, he is not out of danger, as meningitis may supervene as the result of (1) infection from the eustachian tube and middle air spaces, or of (2) contamination of the extravasated blood via the external meatus and tympanic cavity. Meningitis is of course more likely if the labyrinth is involved in the fracture.

Cases of labyrinth injury associated with fracture of the base may be divided into (1) recent and (2) old. (1) In the former cases we have marked hemorrhage into the inner ear. Even in those cases in which the labyrinth capsule is not injured we meet with small hemorrhages in the inner ear and eighth nerve. The auditory nerve may be ruptured and the crura of the stapes torn from the footplate. (2) In old cases the line of fracture may be visible—partly closed by fibrous tissue and partly by new bone. In a case examined by Nager many years after injury there was new connective tissue and bone in the vestibule and in the basal coil of the cochlea. In other cases the line of fracture is not evident.

Case 1.—Male, aged 54, knocked down by a street car. On admission the patient was pale, irritable and restless; pulse 64, temperature 96° F. Bruises were present on the back of the scalp and above the left ear. There was profuse bleeding from the right ear, but no flow of cerebrospinal fluid. (As will be seen later the labyrinth capsule in this case was not injured.) The right pupil was more dilated than the left. Four hours later the patient became comatose and the breathing stertorous. The left arm was found to be flaccid. Operation was performed by a general surgeon. On incising the dura mater blood escaped. The patient died ten hours later.

Postmortem.—The fracture runs along the roof of the middle ear cleft. The middle meningeal artery is lacerated and the roof of the tympanic cavity is splintered. The tympanum and mastoid cells are filled with blood. The fracture extends from the anterior part of the left middle fossa obliquely backwards through the squamous temporal and parietal bones to the lambda, and thence to the right parietal and squamous regions. From here it passes down to a point behind the

right petrous temporal, crosses the sigmoid sinus and then runs along the anterior border of the right petrous bone.

Microscopic Examination of the Right Ear.—The tympanic membrane is ruptured; blood present in tympanum (Figs. 17 and 18); hemorrhage in the region of the geniculate ganglion (Fig. 19); bleeding in the fossa subarcuata. The cochlea shows slight hemorrhages in the modiolus (Fig. 20), but the scalæ of the cochlea are free from blood (Fig. 17). The vestibule and canals show no extravasated blood, but hemorrhage is present in the fundus of the internal meatus (Fig. 19) and in the aqueduct of the cochlea.

Case 2.—Male, aged 41, fell from a scaffold only nine feet high. When found the patient was conscious but was bleeding from the right ear. On admission the face was pale and pulse feeble. There was a flow of blood and cerebrospinal fluid from the right ear. (Microscopic examination showed fracture of the petrous bone involving the labyrinth and extending from the inner wall of the middle ear to the internal auditory meatus.) The patient was very deaf. Coma supervened and the breathing became stertorous. Later general convulsions came on, the eyes being turned to the right—i. e., conjugate deviation to the side of the labyrinth lesion. The right pupil was dilated. Left side of the face and left arm became paralyzed, and the respiration of the Cheyne-Stokes type; pulse 76. An operation was performed by a general surgeon. A fissured fracture was found running from the anterior part of the right squamous temporal downwards and backwards. The bleeding point could not be located. The external carotid was therefore ligatured. The patient died eight hours after operation.

Postmortem.—Skull thicker than normal; carotid arteries thickened; considerable amount of clot present behind the trephine opening. The brain had not expanded. Both lungs showed several gummata. The fracture bifurcated, one limb passing along the roof of the middle ear cleft and the other passing inwards through the labyrinth. In preparing the specimen the outer wall of the middle ear, along with the malleus, incus and tympanic membrane became separated, as they were already loose. Blood was present in the eustachian tube, tympanic cavity, mastoid antrum and cells.

Microscopic Examination of the Right Ear.—The line of fracture passes through the superior and external canal (Fig. 21), downwards through the oval window (Fig. 22), involving the stapes, and below this through the promontory (Fig. 23). The fracture reaches the internal meatus (Fig. 23), but does not extend to the dura covering the posterior surface of the petrous bone. Posterior crus of the stapes is broken. Cochlea: There is marked hemorrhage in the spiral ligament and in the scala tympani and vestibuli, especially in the basal coil (Figs. 22 and 23). There is very little blood in the cochlear canal itself. There are a few small scattered hemorrhages in the modiolus (Fig. 22). The utricle and saccule are ruptured and there is blood in both the peri- and endo-lymphatic spaces of the vestibule (Fig. 22). Semicircular canals: Blood is present in the endolymphatic and perilymph spaces of the superior and external canals (Fig. 21). There is slight hemorrhage along the vestibular nerve, especially along the branch to the saccule. The cochlear nerve to the basal coil also shows hemorrhage (Fig. 22). There is comparatively little hemorrhage present in the internal meatus.

Case 3.—Male, aged 44, laborer. Two days before admission, as he was going home under the influence of alcohol, he slipped and fell on the pavement, striking the right side of his head. He was not unconscious after the accident, but there was bleeding from the right ear and also from the mouth. After the accident the patient suffered from severe vertical headache. On admission he felt as if he were rotating from left to right about a vertical axis. The right ear has been quite deaf since the accident. He has felt sick but has not vomited.

Examination.—Watery discharge from right ear. Right tympanic membrane red and bulging; complete deafness in right ear; Weber lateralized to good ear; spontaneous nystagmus to left (sound side) of second degree; patient tends to fall to the right and shows a pointing error to the right; no facial paralysis. Temperature, 101° F., rose to 103° F. Kernig's sign absent; lumbar puncture: cerebrospinal fluid blood stained but not under pressure. Two days later patient very ill. Severe headache and backache. Temperature 105°, pulse only 72; Kernig's sign present; slight facial paralysis on right

side; second lumbar puncture; fluid under great tension. Polymorphs greatly increased.

Postmortem.—Blood present over the vertex in subdural space and in right temporal region. At the base purulent leptomeningitis was present over pons, medulla and lower surface of cerebellum. Laceration of left temporosphenoidal lobe. The right temporal bone showed a stellate fracture involving both petrous and squamous portions.

Microscopic Examination. External Auditory Meatus.—The anterior wall is fractured (Figs. 25, 26 and 27). The tympanic membrane is generally thickened and is ruptured below and behind the handle of the malleus (Fig. 27). The outer wall of the eustachian tube is fractured. There is a fracture through the roof of the tympanic cavity (Figs. 26 and 27). There is also a fracture of the floor of the tympanum—i. e., of the roof of the jugular bulb (Fig. 25). There is hemorrhage in the outer part of the attic (Fig. 26). In the lower part of the tympanum posteriorly the exudate is purulent (Fig. 26). The stapes shows at least two fractures. The malleus and incus are not fractured. The fracture goes through the facial canal above the oval window and passes through the upper margin of the window. There is also a fracture of the region of the round window (Fig. 25). One can trace the continuity of the exudate in the niche of the round window with that in the scala tympani. Cochlea: Only the intravestibular portion of the cochlea is fractured (Fig. 25). There is hemorrhage in the spiral ligament. Corti's organ is unrecognizable. All three scalæ contain exudate, but the scala tympani shows much more than the others (Fig. 24). Vestibule: The fracture runs into the roof of the vestibule and then downwards through the bony spiral lamina and the region of the round window into the tympanic cavity and finally passes through the floor of the cavity (Fig. 25). The vestibule itself is almost completely filled with hemorrhage. The tip of the promontory is almost chipped off. Canals: There is no fracture of the labyrinth capsule in the region of the canals. The lateral canal contains pus and blood (Fig. 26). The other canals are more normal. Internal meatus: The fracture extends right through from the floor of the middle fossa, through the bony roof and floor of the meatus (Fig. 24), to the region of the opening

of the aqueduct of the cochlea. Some cellular exudate is present in the fundus of the internal meatus (Fig. 24).

Case 4.—Male, aged 6, suffered from fracture of the base of the skull in August, 1913, with bleeding from the right ear. He recovered and remained well for one year, till August 30, 1914, when earache (right ear) and headache came on. August 31, 1914: vomiting, unconsciousness, jerkings of right arm. Rigidity of neck. Kernig doubtful. Temperature 101° F., pulse 110. Death September 1, 1914.

Postmortem (five and a half hours after death).—Flattening of convolutions; cerebrospinal fluid increased and turbid; exudate in interpeduncular space. Sphenoid and ethmoid healthy. Left middle ear contained turbid fluid. Old fracture of roof of right mastoid antrum.

Microscopic Examination of Right Ear.—The mastoid cells contain pus (Fig. 29). There is a gap in the roof of the mastoid antrum filled with fibrous tissue, and there is a piece of bone lying loose in the midst of this fibrous tissue, with pus around it (Fig. 29). This piece of bone proves to be the incus, which had become dislocated backwards into the mastoid antrum. From the appearance of the well formed fibrous tissue surrounding the incus it is evident that this is an old dislocation. (What appears to have happened is that the incus became dislocated backwards at the time of the fracture in 1913, retaining its attachment to the floor of the aditus, and at the same time there was a fracture of the roof of the antrum. The patient recovered at the time, as no infection occurred, but one year later when he developed an acute suppurative otitis media the infection passed by way of the tympanic cavity, the aditus and antrum, through the old gap, which was apparently filled with fibrous tissue, to the intracranial structures, and the result was purulent leptomeningitis and death.) The lining of the eustachian tube is very vascular and swollen, and the submucosa is infiltrated with pus cells. The tympanic cavity contains pus. The footplate of the stapes is quite normal. It is the long process of the incus which has been broken (Fig. 29). The head of the malleus is ankylosed to the new bone which has been formed in the roof of the middle ear (Fig. 28). Cochlea: There are many white cells in the scala tympani in the basal coil, but the pus does not extend as far

as the helicotrema. There is some infiltration of pus cells from the internal meatus into the spiral ganglion of the basal coil. Corti's organ is quite normal. The vestibule and canals are quite healthy. Internal meatus: There are many white cells in the internal meatus within the arachnoid sheath.

4. WAR INJURIES OF THE EAR.

A description of war injuries of the ear written by a surgeon at a clearing station or a hospital near the front would differ very greatly from one written by a specialist attached to a base hospital, and still more from an account penned by an otologist at a military hospital at home. The first would deal mainly with cases of severe wounds of the head involving the ear, due to fragments from bursting shells or bombs or to rifle bullets—injuries which are fatal in a very high percentage of cases. On the other hand, otologists at home see comparatively few of these cases, but have to deal with indirect injuries of the ear caused by explosions or loud noises—i. e., with “shell” or “explosion” deafness, or “noise” deafness.

Wounds of the ear may lead to dangerous intracranial complications. Infected bullets or fragments of shell cause supuration, especially when they carry in infected hair or skin. Secondary infection of the wounds is even more important than primary—i. e., bacteria can find their way to the injured middle ear from the nasopharynx via the eustachian tube or from the external meatus through a rupture of the drumhead.

The Middle Ear.—Rupture of the tympanic membrane is a fairly common war injury, and generally results from the explosion of a shell or bomb close to the sufferer. The lesion may be due to the sudden great increase of pressure which occurs first of all, or to the rapidly following negative pressure. The effect of the explosion varies with its distance from the ear, the size of the shell and the nature of the explosive. Lermoyez says that the effects of explosion are far greater in a trench than in the open field. The tears are often extensive and may give rise to the impression of actual loss of substance. The symptoms are slight bleeding from the ear, followed by serous discharge; there is also deafness and frequently vertigo and pulsating tinnitus. (The latter symptoms are, however, due to a concomitant lesion of the inner ear.) The question of infection is even more important than that of rupture of

the drumhead. Direct wounds of the middle ear always lead to extensive injury—usually to comminuted fractures of the petrous bone. It is only in rare cases that the middle ear alone is injured, and even then suppurative otitis media—usually of long duration—almost invariably follows. Frequently there is extensive splintering of the bone. The tympanic membrane and ossicles are torn or destroyed, and there is extensive hemorrhage into the drum cavity, and antrum. The facial nerve is usually involved. In many cases the inner ear is also involved, and the lesion may extend to the intracranial structures. It rarely happens that shell fragments can remain in the mastoid without causing serious reaction.

The Labyrinth.—War injuries of the labyrinth may be classified as follows: (A) Direct injury due to bullets or fragments of shell or bomb. (B) Indirect injury in fractures of the skull. (C) "Noise" deafness, which is especially seen in artillerymen and naval gunners. (D) "Shell" or "explosion" deafness, due to the bursting of mines, shells or bombs close to the soldier.

(A) Direct Injury of the Labyrinth.—The symptoms of labyrinth injury are well known—deafness, tinnitus, dizziness, disturbance of balancing, vomiting, nystagmus, etc. Fifty per cent of the cases are killed outright at the time of the injury. Haymann states that in the other 50 per cent death occurs later from septic meningitis. The onset of meningitis may be delayed as long as six weeks.

(B) Indirect Injury of the Labyrinth in Fracture of the Skull.—The labyrinth may be indirectly injured by blows on the head from a rifle butt or by shot wounds of the head or face. The symptoms of such injuries are extreme or total deafness, marked disturbance of balancing, with loss of the cochlear and vestibular functions. If the labyrinth be injured there is an escape of cerebrospinal fluid from the ear along with blood. In the cases in which the labyrinth capsule is not injured there is no escape of cerebrospinal fluid; but, nevertheless, deafness results from hemorrhage into, or tearing of, the eighth nerve. On lumbar puncture blood may be present in the cerebrospinal fluid.

(C) "Noise" Deafness.—Wittmaack was the first to show that continuous noise conveyed to the ear by air or bone con-

duction did not cause hemorrhage or tearing of the membranous labyrinth but produced a degenerative neuritis in the cochlear apparatus. The vestibular apparatus showed no change. According to Wittmaack, the primary changes occurred in the spiral ganglion and nerve, while those in Corti's organ were only secondary. Yoshii, on repeating Wittmaack's experiments, found that Corti's organ was affected first of all, and that the degenerative changes in the cochlear ganglion and nerve followed later. Both von Eicken and Hoessli came to the conclusion that air conduction of sound was much more effective than bone conduction in producing injuries of the inner ear. After prolonged exposure to the sound of rifle and gun fire, deafness is usually marked and tinnitus not uncommon.

(D) "Shell" or "Explosion" Deafness.—The pathology of explosion or shell deafness is still very vague. (a) Some otologists believe that there are changes in the membranous labyrinth of a molecular nature leading to degenerative neuritis affecting the nerve endings, ganglia, etc. (b) Others hold that the membranous labyrinth is torn and that hemorrhages occur. Yoshii found that explosions produced close to the ears of animals caused rupture of the drumhead and hemorrhages in the cochlear and vestibular apparatus. Hoessli caused explosions at a greater distance from the animals and did not find rupture or hemorrhage but noted changes in Corti's organ, the cells of which were shrunk and flattened. (c) Zange has experimented on animals, but has found no changes in the labyrinth. He therefore believes that the condition is due to small hemorrhages in the pons, medulla and cerebellum.

WAR INJURIES.

Case 1.—The case was one of shrapnel injury of the left ear followed by suppurative otitis media. When the patient was admitted to the Royal Infirmary, Edinburgh, about a month after the injury, symptoms of cerebellar abscess were present. Operation, performed by a general surgeon, revealed mastoiditis and small metallic particles in the mastoid antrum. In spite of the evacuation of the cerebellar abscess the patient died. The autopsy showed early meningitis and sinus thrombosis in addition to the cerebellar abscess. Microscopic exam-

ination of the ear demonstrated a thickened, infiltrated and perforated drumhead (Figs. 31 and 32), with fracture of the malleus. The lower part of the handle was separated from the drumhead and drawn inward by the tensor tympani (Fig. 32). The mucosa of the middle ear was distinctly swollen (Fig. 33), and the tympanic cavity, antrum and cells were full of pus. The joint between the head of the malleus and the body of the incus was dislocated (Fig. 30). The incudo-stapedial joint was healthy, but there was a fracture of the footplate of the stapes and early invasion of the vestibule through the annular ligament (Fig. 32). The cochlea showed hemorrhage in the scala tympani of the basal and middle coil and in the opening of the perilymphatic aqueduct (Fig. 33). but the nerve apparatus of the cochlea appeared almost healthy. The neuroepithelium of the saccule and utricle and of the cristæ of the canals was desquamating. The internal meatus showed meningitis (Fig. 33).

Note.—Several of the changes noted in this case may have been due to small pieces of shrapnel causing—e. g., fracture of the malleus and stapes, hemorrhage in the scala tympani and fracture of the bony capsule of ampulla of posterior canal (Fig. 5).

Case 2.—Indirect injury of the ear, due to bullet wound of the parietal ganglion. The patient was hit in the fronto-parietal region, and the bullet came out just above the ear on the same side. There was no apparent injury of the ear itself, but the parietal bone was shattered and the brain lacerated.

Middle Ear Spaces.—Free blood is present in the lower part of the tympanic cavity, in the niches of the round and oval windows (Fig. 35) and in the mastoid antrum. Labyrinth contents: The nervous structures of the inner ear appear to be normal, but a little blood is present in the aqueduct of the cochlea, near its cranial end, and also around the vestibular nerve to the utricle. Internal meatus: There is a considerable amount of hemorrhage within the arachnoid sheath which surrounds the seventh and eighth nerve (Fig. 34).

Case 3.—High explosive shell injury. This soldier was taking cover in a house when a high explosive shell hit the house and burst into the room in which he was.

The patient was wounded in many places. On admission to the Casualty Clearing Station the patient was found to be semiconscious, with subnormal temperature, small rapid pulse, cold clammy skin and multiple small wounds. In fact, he showed the usual signs of high explosive shell injury, including marked deafness. This case shows certain definite changes—viz., rupture of the tympanic membrane (Fig. 36), slight hemorrhage in the middle ear spaces and in the fundus of the internal auditory meatus. The structures of the membranous labyrinth show normal conditions.

Case 4.—Injury to the ear due to the bursting of a rifle grenade. The grenade burst close to the left side of this soldier and caused extensive general injury. (Rifle grenades, though much smaller than shells, contain a very powerful form of high explosive.—J. Fraser.) The changes in this case of ear injury due to grenade explosion are fairly definite and include a recent rupture of the tympanic membrane, the edges of which are partially glued together by fibrin (Fig. 39). Further, there are hemorrhages in the tympanic cavity, in the marrow spaces in the roof of this cavity, around the geniculate ganglion and in the fossa subarcuata. There is also bleeding in the scala tympani of the cochlea in the region of the round window (Fig. 39). There is a marked hemorrhage in the fundus of the internal meatus. The otolith membranes of the saccule are separated (Figs. 37 and 38), and the neuroepithelium is irregular, while the cupula of the lateral canal is also separated (Fig. 40). Although the outline of Corti's organ is preserved, the hair cells and some of the supporting cells cannot be made out, so that the organ appears ghostlike.

Case 5. Shell Explosion.—The patient suffered from severe concussion, and the right side of the head and body presented multiple wounds. The changes in this case are very slight. This may possibly be explained by the presence of a plug of wax in the external meatus. There are small hemorrhages in the tubal part of the tympanic cavity, in the canal for the tensor tympani, in the cranial ends of the fossa subarcuata and perilymphatic aqueduct and in the fundus of the internal meatus. The neuroepithelium of the labyrinth shows little change, but the detachment of the otolith membrane of the

utricle and of the cupulæ of the lateral and superior canals is worth recording.

Case 6.—Injury by high explosive shell. This soldier caught the full blast of a high explosive shell, which burst close to his left side and shattered this side from head to foot. In this case the force of the explosion seems to have been expended in rupturing the drumhead (Fig. 41), so that the structures of the inner ear along with the nerves in the internal meatus have escaped. Owing to a fault in the first embedding of the specimen, air was present in the hollow spaces of the labyrinth, and it is therefore unwise to be dogmatic as to the condition of the membranous labyrinth. The rupture of the sacculæ and of portions of the membranous cochlea are probably artefacts produced by the use of the suction apparatus. The rupture to the posterior part of the annular ligament may also be due to the same cause.

Summary.—In four cases of "shell" or "explosion" deafness (Cases 3, 4, 5 and 6), the changes are as follows: (a) Rupture of the drumhead in Cases 3, 4 and 6. (In case 5 a large plug of wax was present in the external meatus, and probably for this reason the tympanic membrane was not injured.) (b) Hemorrhage in the middle ear spaces in all four cases. (c) Slight hemorrhage in the scala tympani in the region of the round window and basal coil of the cochlea (Case 4). (d) Slight degenerative neuritis of the cochlear apparatus—e. g., loss of hair cells, slight flattening of the acoustic papilla and disappearance of some of the supporting cells, especially in Case 4; shrinkage of the ganglion cells of the spiral ganglion; uneven staining of the cochlear nerve. (All these may be post-mortem changes.) (e) Displacement of the otolith membrane of the sacculæ and utricle (Cases 4 and 5), artefacts? (f) Displacement of the cupula of one or more of the canals (Cases 4 and 5). (g) The vestibular apparatus, as was to be expected, showed less change than the cochlear. The neuroepithelium of the sacculæ and utricle and the cells of the vestibular ganglion in the internal meatus appear to be better preserved than the corresponding parts of the cochlear apparatus. (h) Hemorrhage was observed in the fundus of the internal meatus at the points where the nerves enter the bony canals (Cases 3, 4 and 5).

5. LABYRINTHITIS.

Acute inflammatory affections of the labyrinth may be classified in various ways: thus, inflammation of the inner ear may be described as circumscribed or diffuse. Further, the diffuse form may be divided into stages. First of all, there is a serous, fibrinous or hemorrhagic stage. Later, the exudate in the labyrinth becomes purulent. The purulent stage is followed by the formation of granulation tissue in the hollow spaces of the inner ear—the granulations arising from the endosteum. Finally, the granulation tissue is organized into fibrinous connective tissue, in which more or less new bone formation occurs. Sometimes the process of ossification is so extensive that the whole of the hollow spaces of the inner ear are entirely obliterated.

Labyrinthitis may also be classified according to the causal disease or organism—e. g., labyrinthitis due to scarlatina, measles, influenza, etc. Further, labyrinthitis may arise either from acute or from chronic middle ear suppuration. In the former case the path of infection is almost invariably through the oval or round windows, whereas in the latter the labyrinth is usually invaded by the erosion of the prominence of the lateral semicircular canal by cholesteatoma formation. To put it in another way, in acute purulent otitis media the labyrinthitis is a diffuse one from its onset, whereas in chronic middle ear suppuration associated with cholesteatoma the labyrinthitis is first of all circumscribed and may remain so for a considerable period. An exacerbation of the middle ear suppuration, however, may lead to rapid spread of the circumscribed labyrinthitis, which thus becomes diffuse.

In order to illustrate the various types of simple inflammatory affection of the labyrinth it will be best to give a short account of the case history of the patients, followed by a description of the appearances found on microscopic examination of the inner ear after death.

CIRCUMSCRIBED LABYRINTHITIS.

Case 1.—George D., male, aged 4 years. Chronic middle ear suppuration (right) for three years, since scarlet fever. Pain in right ear, shivering, dizziness and vomiting for three days. Examination: Foul smelling discharge; red swelling behind right ear; mastoid tenderness; Rombergism marked. Tem-

perature 99. Tongue dry and furred. Scar of old gland abscess in neck. Watch heard on both mastoids. Conversation voice at two feet (R.), with finger closure of left ear. Nystagmus to side of lesion only. Operation on October 14, 1913. Mastoid cortex grayish white; large foul smelling abscess in mastoid, with extradural, perisinus abscess; dura yellowish gray and sloughy; sinus not opened; antrum full of cholesteatoma. Wound left open. Breathing stopped on one occasion during operation but restarted spontaneously. On the following day, temperature 101 at 8 p. m. Slight vomiting. Cerebrospinal fluid under pressure and contained polymorphs and organisms. Second operation on October 17th. Sinus further exposed, slit up and found thrombosed. Dark red clot; bleeding obtained from both ends. Jugular not ligatured; incision in cerebellar dura; gauze drain introduced. On following day Kernig's sign positive; child restless; early optic neuritis. Exploration of cerebellum without anesthetic; evacuated foul smelling cerebellar abscess. Urotropin given. On October 27th, vomiting present; nystagmus to side of lesion. No reaction in wound. Coma supervened. Temperature 104. Death on October 31st. Postmortem: General purulent meningitis. Large cerebellar abscess. Right internal jugular not thrombosed.

Microscopic examination of right ear: Round window niche largely filled up by connective tissue; cholesteatoma on inner wall of tympanum; granulations in roof of tympanum; caries of inner wall of attic. Erosion of bony wall of lateral canal (Fig. 42). Cochlea almost normal. A little pus in perilymphatic aqueduct. Purulent infiltration of modiolus. Utriculus and sacculus normal. Circumscribed labyrinthitis in lateral and posterior canals. In the latter there is new connective tissue formation with pus cells. The extradural abscess had evidently extended behind the labyrinth. Meningitis in internal meatus. Thrombus in jugular bulb.

Case 2.—Donald H., male, aged 23. Chronic middle ear suppuration (bilateral). Pain in right ear. Vomiting for two weeks, with shivering. Patient admitted to medical ward with diagnosis of "biliary colic." Rigor on day of admission (June 16, 1918). Temperature 103, pulse 116, respiration 36. Slight delirium.

Tongue dry and furred. Head retracted. Kernig present. Nystagmus to both sides. Tenderness of right mastoid. Patient not completely deaf in right ear. Immediate operation. Cerebrospinal fluid turbid and under tension (diplococcus). Pus under pressure in antrum. Sinus healthy. Next day, temperature 102. Rigor. Second operation: Right internal jugular ligatured. Next day patient restless; severe headache; heroin given. June 19th. Lumbar puncture. Cerebrospinal fluid under great tension and almost purulent; films show many organisms. Fever continued, with rigors. Coma supervened. Death June 23rd. (Patient had an uncle who died from brain abscess.) Postmortem: Basal meningitis. Temporosphenoidal abscess present, with thick walls. Abscess had ruptured into lateral ventricle. Leakage into lateral ventricle had probably occurred before admission. Sigmoid sinus appeared healthy and therefore the rigors were probably due to meningitis. (Blood cultures negative.)

Microscopic examination of right ear: Cholesteatoma in tympanum, with thickened mucosa; new bone formation in roof of aditus and antrum; bony wall of lateral canal eroded (Fig. 43). Granulation tissue on inner wall and roof of antrum reached middle fossa—i. e., route of infection of brain abscess; engorgement of marrow spaces in floor of middle fossa, with absorption of bone; some marrow spaces contain pus; cochlea normal except for a little pus at opening of perilymphatic aqueduct. Slight cellular exudate in vestibule on inner aspect of oval windows. Lateral canal shows circumscribed labyrinthitis. Meningitis in internal meatus.

CIRCUMSCRIBED FOLLOWED BY ACUTE PURULENT LABYRINTHITIS.

Case 3.—Annie B., female, aged 15 years. C. O. M. S. (right). Noises in right ear and giddiness for one month; earache for one week; headache and pain in neck for five days; vomiting for four days; shivering two days. Patient drowsy on admission; slightly cyanosed. Temperature 98.6. Tongue furred and dry. Right ear quite deaf with noise box in left ear. Weber lateralized to better ear; slight spontaneous nystagmus to left. Caloric reaction absent in right ear but fistula symptom present. Cerebrospinal fluid clear but under increased pressure. Kernig present; stiffness of neck; no optic neuritis.

Immediate operation: Foul perisinus abscess containing

gas; sinus wall sloughy; cholesteatoma in antrum; ossicles absent; fistula in lateral canal; sinus thrombosed; free bleeding obtained from torcular end; right internal jugular ligatured. At this point pulse very feeble. Labyrinth operation not performed. Saline injections given. During next week rigors continued and optic neuritis developed. Operation performed on jugular bulb. Death seventeen days after first operation. Postmortem: No reaction in wounds. Clot in right lateral sinus and also in superior longitudinal and straight sinuses. No meningitis or abscess of brain. Infarcts present in lungs.

Microscopic examination of right ear: There is rarefaction of the bone in the roof and floor of the tympanic cavity. Cochlear canal full of pus, whereas the scala vestibuli and scala tympani contain only fibrinopurulent exudate. Aqueduct of cochlea contains pus. The vestibule contains curdled lymph. Fistula present in lateral canal (Fig. 44); lumen of canal showed pus becoming organized. Other canals show purulent contents. Almost no meningitis in the internal meatus. The clot in the jugular bulb is becoming organized.

SEROUS LABYRINTHITIS.

Case 4.—John F., male, aged 11 years. C. O. M. S. (bilateral) for six years. Earache, headache and irritability for six days before admission. On examination, temperature 100; right mastoid tender; granulations and pus in right meatus; conversation voice at one foot (right ear); perforation of Shrapnell's membrane on left side; whisper at one foot (left); no nystagmus; cerebrospinal fluid clear and not under pressure; no photophobia. Three days after admission, temperature rose to 105°; head retracted; sudden facial paralysis on right side. Operation: Right mastoid contained cholesteatoma; sinus wall healthy; right temporosphenoidal lobe explored with negative result. Cerebrospinal fluid turbid on lumbar puncture; death two days after operation. Postmortem: Purulent meningitis at base; no brain abscess; venous sinuses healthy.

Microscopic examination of right ear: Cholesteatoma and polypus present in middle ear; bony exostosis from promontory; cochlea normal; vestibule healthy; slight serous labyrinthitis in lateral canal (Figs. 45 and 46); other canals normal. Meningitis in internal meatus, with slight hemorrhage and engorged vessels.

ACUTE SUPPURATIVE OTITIS MEDIA, PURULENT LABYRINTHITIS
AND LEPTOMENINGITIS WITHOUT RUPTURE OF THE
TYMPANIC MEMBRANE.

The following case shows that in a case of acute middle ear suppuration the pus in the tympanic cavity may penetrate into the labyrinth through the round and oval windows and from the labyrinth may infect the subarachnoid space before the purulent exudate in the tympanic cavity bursts through the tympanic membrane. John J., aged 12 years, was quite well till the morning of March 17, 1913, when he refused breakfast on account of severe earache and slight frontal headache. The boy vomited at 11 a. m. His doctor was called in and noted choreic movements of the hands. Vomiting continued at short intervals until the following morning. After this it was not so constant. The patient slept on and off during these first days of illness; he had frequent attacks of restlessness, and screamed if he were touched. At 1 p. m. on the day of admission (March 20th) he lapsed into a semicomatose condition.

Family History.—Father died of consumption at age of 36. One brother, aged 18, committed suicide three weeks before patient's illness began; this brother, who suffered from tubercular cervical adenitis, had been despondent for some time on account of severe deafness due to chronic suppurative otitis media.

Examination by Physician in Medical Ward (the boy was not seen by an otologist).—Temperature 102.4° F.; pulse 94. Conjugate deviation of eyes to the left. He moans constantly but there is no distinct cry. Kernig's sign present; no discharge from the ears; both tonsils large and red; abdomen slightly tense but not scaphoid. Lumbar puncture yielded milky fluid under considerable pressure. (Films showed many polymorphs and numerous capsulated diplococci, some in short chains; pneumococci on culture.) Next day Cheyne-Stokes respiration and death.

Postmortem.—Convulsions flattened; pus in subarachnoid space over both sides of cerebral cortex. Mastoid cells on both sides filled with pus. The left internal auditory meatus was also full of pus. On the right side the middle ear spaces contained pus. Ethmoid and sphenoid sinuses congested, but contained no pus. Hemorrhages in the pleura; bronchial

glands enlarged; on section right lung showed intense congestion and small congested collapsed areas. The mucous membrane of the trachea and large bronchi covered with blood stained seropurulent fluid.

Microscopic Examination.—Right ear showed otitis media but only hemorrhage in labyrinth (early stage of labyrinthitis) (Fig. 47). Eustachian tube and tympanic cavity full of pus (Fig. 50). The annular ligament is intact and is not infiltrated with pus cells (Fig. 49). The secondary tympanic membrane is moderately thickened but is not infiltrated (Fig. 51). Cochlea: Hemorrhage in the scala tympani and scala media in the neighborhood of the round window (Fig. 51). The cochlear opening of the perilymphatic aqueduct contains pus which has apparently found its way up the aqueduct from the subarachnoid space (Fig. 51). Hemorrhage is present in the osseous spiral lamina of the basal coil (Fig. 48). The modiolus and the nerve canals are infiltrated with pus, which appears to have invaded from the subarachnoid space of the internal meatus. There is a large hemorrhage in the perilymphatic space of the vestibule (Fig. 49). Internal meatus: Pus is present and surrounds the cochlear and vestibular nerves (Fig. 47).

Left ear: The lining membrane of the eustachian tube is swollen and the submucous tissue densely infiltrated with small cells. Tympanic cavity filled with pus (Figs. 52 and 55); the mucous membrane over the promontory is greatly thickened to form a long slender polypus. The posterior part of the tympanic membrane bulges outwards towards the meatus. The epidermic layer is desquamating and the fibrous layer is disintegrated by small cell infiltration (Fig. 54). Pus cells form a small abscess just beneath the desquamating epidermic layer (myringitis). At this spot rupture of the membrane was just about to occur. The footplate of the stapes is normal, but the anterior part of the footplate is tilted outwards. The annular ligament is infiltrated with pus cells, which are finding their way through it into the vestibule (Fig. 53). All coils of the cochlea contain pus (Fig. 52). The cochlear duct contains pus, and Corti's organ is disintegrated. The modiolus is infiltrated with pus cells and the blood vessels are greatly dilated. Round window: The secondary tympanic membrane

is greatly swollen and infiltrated with pus cells, which may be seen making their way through it into the scala tympani (Fig. 56). Vestibule: There is a considerable collection of pus just internal to the footplate of the stapes (Fig. 53).

ACUTE PURULENT LABYRINTHITIS; INFECTION THROUGH
OVAL WINDOW.

Case 6.—Mrs. C., female, aged 20, gave a history of chronic suppurative otitis media (bilateral) since the age of 2 years. She had had pain in the right ear for six weeks before admission and vomiting for one week, with shivering. There was no dizziness. The patient was first seen on October 13, 1908, when it was noted that she looked ill and emaciated. The temperature was 102° F., and there was mastoid tenderness on the right side. The patient complained of pain in the occipital region and down the spine. The knee jerks were exaggerated. Next day facial paralysis (right) was present and third degree nystagmus to left; nystagmus also present on looking upwards. There was no ocular paralysis and the fundi were normal; knee jerks absent; grasp of right hand weaker than left.

Operation.—Mastoid cells and lateral sinus healthy; antrum contained granulations; dura internal to sinus showed granulations and softening; oval window empty; lateral canal healthy. Death occurred one day after operation. Postmortem: Basal meningitis.

Microscopic examination of right ear: Mucosa of middle ear greatly thickened and infiltrated, especially round windows (Fig. 57); fibrinous exudation in all scalæ, with few pus cells; basal coil most affected (Fig. 59); modiolus not infiltrated. Stapes displaced into vestibule (Fig. 57) and pus present here; oval window empty (Fig. 58). Empyema of saccus endolymphaticus. Purulent infiltration of canals. Meningitis in internal meatus, with hemorrhage.

SUBACUTE LABYRINTHITIS LATENT.

Case 7.—Thomas G., male, aged 17 years, was admitted to a general surgical ward on September 5, 1911. He had suffered from chronic suppurative otitis media, right, since measles in childhood. Three days before admission the discharge suddenly ceased and pain in right ear commenced. On following

day swelling appeared behind the ear and the patient had a sudden attack of giddiness.

On examination (September 6th): Abscess over mastoid; temperature 100.4° F., pulse 100; sagging of posterior wall of the meatus.

The radical mastoid operation was performed. The patient was discharged from hospital one month after operation but was readmitted seventeen days later in a semicomatose condition. His face was pale; temperature 96° F.; pulse 68; pupils dilated and equal; they did not react to light. The patient lay curled up on the right side; incontinence of urine; almost stertorous breathing. The operation cavity was found to be full of pus. One hour after readmission the patient suddenly became flushed and perspired freely; pupils contracted; respiration gradually stopped but the heart continued to beat. Artificial respiration was carried out but the pulse became feeble and patient died. Postmortem: Abscess in right lobe of cerebellum.

Microscopic examination of right ear: Small celled infiltration of submucosa of middle ear and new bone formation on promontory and in attic (Fig. 61); oval window not perforated; polypus projecting from inner wall in region of lateral canal (Fig. 60) and hanging down into the tympanic cavity and external meatus. This polypus contains an abscess and communicated with a fistula in the lateral and posterior canals and also with an extradural abscess in the posterior fossa (Fig. 61). The polypus is covered with squamous epithelium except at the tip, where it is ulcerated. There is extensive softening of the labyrinth capsule, especially in the region of the canals (Fig. 61). The hollow spaces of the cochlea show curdled lymph and a little pus. There is also some new connective tissue formation in the scala tympani and scala vestibuli of all coils. The scala media is dilated and the spiral ligament atrophied. The vessels of the modiolus are distended. The spiral ganglion is atrophied and there is new connective tissue formation in the spiral canal. The saccule is dilated and contains pus and coagulum. The utricle is difficult to recognize. The perilymph space of the vestibule contains pus, which is becoming organized into granulation and connective tissue. There is even a little new bone formation next

to the endosteum. The canals contain pus, which is becoming organized into granulation tissue. The posterior canal shows slight new bone formation. The internal meatus shows small celled infiltration and also new formation of connective tissue. The vessels here are dilated. An extradural abscess is present in the region of the saccus endolymphaticus.

PANLABYRINTHITIS (I. E., PARA-, PERI- AND ENDO-LABYRINTHITIS).

Case 8.—Christina G., female, aged 6, had discharge from the left ear following an attack of measles at the age of 3 years. On December 25, 1911, she had severe pain in the left ear followed by vomiting, dizziness and headache (first attack of labyrinthitis). (This fact was not ascertained till after the death of the patient.) These symptoms gradually improved, and by January 8, 1912, she was fairly cheerful. When seen for the first time on January 13 the left external meatus contained pus and granulations; no mastoid tenderness. January 15, 1912. Adenoids and tonsils were removed. Unfortunately the granulations in the left ear were also curetted—a procedure which should be strictly avoided unless the labyrinthine function has been tested and found normal. January 17: Patient cyanosed and vomiting; temperature 103.2° F., pulse 160. January 18, complete deafness in left ear; dizziness present; rotatory and horizontal nystagmus to right (sound side); pulse feeble; tongue furred; diarrhea; cerebrospinal fluid under increased tension, but clear. (Films show streptococci and bacilli.) Operation considered hopeless. January 19, antistreptococcus serum given; facial paralysis present on left side; death.

Postmortem: Cloudy swelling of kidneys, heart and liver; petechial hemorrhages in lungs; enteritis; mesenteric glands showed recent acute inflammation in addition to chronic enlargement; no obvious meningitis. The appearances were those of acute septicemia.

Summary.—The case appears to have been one of septicemia and malignant serous meningitis following the removal of tonsils and adenoids and curettage of ear granulations in a patient suffering from latent labyrinth suppuration.

Examination of Left Ear.—Middle ear: Large perforation of the tympanic membrane (Fig. 65). Tympanum, aditus and

antrum lined by cholesteatoma (Figs. 62 and 65). There is caries of the bone beneath the cholesteatoma. On the inner wall of the attic there is a polypus just external to the ampullated end of the superior canal (Fig. 62). The malleus and incus have been displaced backwards (Figs. 64 and 65) (curetage). The head of the stapes is absent and the footplate necrotic and displaced. The annular ligament and the secondary tympanic membrane are swollen and infiltrated. Labyrinth capsule: There is extensive replacement of the cartilage bone of the labyrinth capsule by granulation tissue beginning in the inner wall of the attic. The disease has spread between the endosteal bone and the lamellar bone (Figs. 62 and 65). There are numerous osteoclasts in the granulation tissue. In the promontory the inflammatory process is invading the bone from the submucous tissue (Fig. 63). Cochlea: The basal coil shows delicate connective tissue and a little new bone in the scala tympani and vestibuli; there is great dilatation of the cochlear duct (Fig. 64). A trace of Corti's organ can be seen in the middle coil; elsewhere it is absent. The capsule of the cochlea shows several areas of "ostitis vasculosa," especially in the region of the anterior margin of the oval window (Fig. 63). The vestibule contains much pus (Figs. 63, 64 and 65). This is especially thick just internal to the oval window. The capsule of the vestibule looks as if it had been dissolved and replaced by granulation tissue. There is necrosis of the bony partition between the internal meatus and vestibule (Fig. 64). The membranous structures of the vestibule cannot be recognized. The canals show much the same changes. The granulation tissue around the posterior canal is in contact with the dura of the posterior cranial fossa. Internal meatus: Pus and granulation tissue are seen along the branches of the vestibular nerve (Figs. 63 and 64). The cochlear nerve is more normal. There appears to have been abscess formation in the posterior part of the meatal fundus, followed by the formation of granulation tissue, and probably by interference with the blood supply of the bony capsule of the vestibule and canals. This would favor the spread of otitis (paralabyrinthitis).

From the clinical history it is evident that the patient had an attack of labyrinthitis on December 25, 1911. When seen for

the first time on January 13, 1912, the inner ear had passed into the stage of latent labyrinth suppuration. The paralabyrinthitis appears to have originated on the inner wall of the attic and was due to curettage of the aural polypus. The child probably died from septicemia due to infection following the operation on the fauces and pharynx in such a septic case.

SPONTANEOUS CURE OF LABYRINTHITIS.

Case 9.—Walter P., male, aged 42. C. O. M. S. (left) since childhood. Loss of appetite and mental dullness for three weeks. Earache and dizziness for three days before admission to Workhouse Hospital. Headache on admission and tendency to fall backwards. Patient transferred, on account of irrational behavior, to the Royal Edinburgh Asylum on May 29, 1912. Tendon reflexes exaggerated on both sides, especially on left. General condition gradually improved. Wassermann reaction negative in blood and cerebrospinal fluid. On July 12, however, patient became confused and irritable. Death three days later. Postmortem: Acute endocarditis. Pachymeningitis externa over roof and posterior surface of left temporal bone. No brain abscess. The pathologist regarded endocarditis as probably secondary to the aural sepsis.

Microscopic examination of left ear: Extradural abscess above and behind labyrinth, affecting both middle and posterior fossæ (Fig. 67). Left drumhead retracted and adherent to inner wall of tympanum (Fig. 67). Drumhead shows small perforation in posterior part. Tympanic cavity almost obliterated. There is dense, small celled infiltration of submucosa over the promontory. Fistula present into basal coil of cochlea. There is some new bone formation in the wall of the tympanum. Head of malleus eroded; incus absent; stapes normal but embedded in thickened mucosa; oval window not perforated; niche of round window entirely filled up by connective tissue (Fig. 67). The hollow spaces of the labyrinth are almost entirely obliterated by connective tissue and new bone formation. There is pus present in the region of the crus commune, which communicated with the extradural abscess. This abscess extends over the convexity of the superior canal and has markedly eroded the bone in this region (Fig. 67). The extradural abscess also communicated by a band of fibrous tissue with the jugular bulb, which, however,

was not thrombosed. The middle and apical coils of the cochlea are less severely affected than the basal coil (Fig. 66), which is entirely filled up by new bone. The scala media of the middle coil is dilated and Corti's organ has disappeared. The scala tympani and scala vestibuli show some new fibrous tissue and bone formation. Further, the opening of the perilymphatic aqueduct is filled up by new bone. The nerve canals of the modiolus are filled up with new fibrous tissue; nerves and ganglia absent. Vestibule largely filled up by fibrous tissue (Fig. 67). Utricle, saccule and endolymphatic aqueduct not recognizable. The canals are less affected than other parts, but the perilymphatic space contains pus and some new formed fibrous tissue. Dura of internal meatus greatly thickened (Fig. 66). Abscess present in floor of meatus. The extradural abscess is in contact with the dura of the roof of the meatus. The seventh and eighth nerves are incorporated in fibrous tissue. Only traces of the vestibular ganglion can be seen.

Case 10.—William N., male, aged 26 years, had suffered from chronic suppurative otitis media (left) since childhood. He was admitted to a general surgical ward with symptoms of cerebellar abscess. The cerebellum was explored behind the sigmoid sinus, with negative result. Ten days later the patient had a rigor, and at the second operation the sigmoid sinus was found to be thrombosed. The jugular vein was ligatured but the radical mastoid operation was not performed. The patient made a temporary recovery. Later he was readmitted on account of ear discharge and sent over to the Ear and Throat department. Examination on admission showed slow pulse and subnormal temperature. The patient was drowsy but did not vomit and had no headache; no tenderness over skull; no sensory aphasia; no paralysis; marked Rombergism; complete deafness on the left side; spontaneous nystagmus to right (sound) side; caloric reaction absent on left side but prompt response obtained on right side.

Third Operation (radical mastoid).—Cholesteatoma in middle ear spaces; lateral canal showed no fistula but had lost the usual white appearance. Labyrinth operation not performed. Eleven days later nystagmus still present but no vomiting. On fifteenth day after the third operation headache and vomit-

ing were present; optic discs normal; cerebrospinal fluid clear. The patient died suddenly next day. Postmortem: Cerebellum adherent to dura in triangular area; abscess in left lobe of cerebellum, with surrounding edema; no meningitis.

Microscopic examination of left ear: Cholesteatoma in attic and aditus; stapes displaced into vestibule (Fig. 69)—probably by contraction of fibrous tissue; round window membrane thickened (Fig. 69); labyrinth capsule very vascular. The hollow spaces of the inner ear are filled up by connective tissue and new bone (Fig. 68). There is, however, still some pus (abscess) in the center of the vestibule (Fig. 70). The membranous labyrinth has disappeared. The internal meatus shows the remains of the seventh and eighth nerves surrounded by vascular fibrous tissue and old hemorrhage (Fig. 68). The jugular bulb contains organized clot.

Case 11.—William A., male, aged 5 years, had had discharge from right ear for more than one year; a large mastoid swelling for two days. Examination showed sagging of the roof of the meatus on the right side. The right ear was quite deaf when the noise apparatus was going in the left. There was no spontaneous nystagmus. Cold syringing of the right ear produced no reaction. Temperature 100.6° ; opacity of right cornea. The patient had been attending a tuberculous hospital.

Operation.—Pus in antrum; roughness of prominence of lateral semicircular canal but no fistula. On curetting the promontory the small spoon passed into the vestibule, which was therefore freely opened up. Dura of posterior fossa exposed and attempt made to open up posterior and lateral canals; these, however, could not be found. The facial nerve twitched on two occasions. On the following day facial paralysis was noted; Kernig's sign present; temperature 103° F.; nystagmus to side of operation; photophobia. Sedatives were given. Later there was marked retraction of the head and opisthotonus; scaphoid abdomen. Death occurred four days after operation. Postmortem: Caseous bronchial glands; large hemorrhage in left parietal region (side opposite to operation); basal meningitis; left mastoid healthy.

Microscopic examination of right ear: Complete filling up of hollow spaces of labyrinth by new bone formation (Fig. 71). There is evidence of bony softening in region of ves-

tibule and basal coil of cochlea, in the spot where the spoon entered at the operation.

Case 12.—Margaret W., female, aged 52. Patient gave a history of chronic suppurative otitis media (right) for over twenty years. Two weeks before admission she had pain in the right ear and four days later a rigor and vomiting. The patient's doctor suspected typhoid.

On examination, no headache or mastoid tenderness; temperature 105° F. Operation: Pus and cholesteatoma in antrum; sigmoid sinus brownish black; fetid pus escaped; free bleeding obtained from torcular end. Internal jugular vein exposed and found to be collapsed; apparently healthy clot reached just above the clavicle. Progress: No reaction in wound; rigors recurred, with vomiting. Blood culture showed streptococcus and antistreptococcus serum was given. Death occurred fourteen days after operation. Postmortem: Septic thrombus in lower end of internal jugular vein; infarcts and abscesses in lung.

Microscopic examination of right ear: Mucosa of middle ear thickened and engorged; cholesteatoma present; some new bone formation on promontory; annular ligament intact but stapes footplate eroded (Fig. 73); bone around labyrinth capsule very vascular; granulation tissue present between posterior canal and posterior fossa (extradural abscess); cochlea entirely replaced by bone (Fig. 72); round window cannot be recognized though the niche can be seen; vestibule shows great dilatation of saccule and utricle (Fig. 73); neuroepithelium of utricle present; new connective tissue and a little bone formation in perilymph space of vestibule. There is evidence of an old fistula in the lateral canal; connective tissue present in crus commune and in other parts of the canals. Internal meatus shows small hemorrhage along the vestibular nerve; cochlear nerve completely atrophied (Fig. 72); facial nerve normal.

6. TUBERCULOSIS OF THE MIDDLE EAR AND LABYRINTH.

Note.—The following remarks on the clinical aspect of tuberculous disease of the ear have been abstracted from an article published by Dr. Logan Turner, *Journal of Laryngology, Rhinology and Otology*, Vol. XXX, 1915, page 209:

Age Incidence.—Of 60 cases, 51 were children and 9 adults.

During the period 1907-14 the cases of middle ear suppuration under fifteen years of age, seen at the Ear and Throat Department of the Royal Infirmary, Edinburgh, numbered 1,797, of which 51, or 2 per cent, were tuberculous. Among these 1,797 cases there were 505 cases of middle ear suppuration under five years and, of these, 48 were tuberculous—i. e., 9 per cent. Under the age of two years there were 172 cases of middle ear suppuration, of which 47 were tubercular—27 per cent. In the first year of life there were only 86 cases of middle ear suppuration, and 43—i. e., 50 per cent—were tuberculous. In 32 of the 43 infants there was evidence of the disease in the first six months of life.

Etiology (in infants and young children).—The majority of the children suffering from tuberculosis of the ear were "bottle fed," and it was most exceptional to obtain any history from the mother of the milk having been boiled or sterilized. With regard to breast fed babies it was a common experience to hear that the breast feeding was supplemented by bottle milk. Mitchell has examined 406 samples of mixed milk collected from the same number of milk shops in Edinburgh; in 82 samples, or 20 per cent, tubercle bacilli were found. (In every instance the inoculation of guinea pigs was the method employed for the detection of tubercle bacilli.) Further, 72 consecutive cases of tuberculous cervical glands in children were investigated by Mitchell. The bovine type of the tubercle bacillus was present in 65 instances (90 per cent) and the human bacillus in only 7 (10 per cent). Eighty-four per cent of the children had been fed on unsterilized cow's milk since birth. John Fraser has examined 70 cases of tuberculous bone disease and found in 60 per cent of the cases the bovine strain of tubercle bacillus.

The second type of tubercular otitis media occurs in the advanced stages of phthisis pulmonalis. St. Clair Thomson found only two cases among 700 patients suffering from pulmonary tuberculosis. Herzog found chronic purulent otitis media in 17 out of 100 cases of "consumption"; 5 of the 17 cases showed labyrinthine involvement. Turner and Fraser have examined 5 cases of purulent otitis media occurring among 120 cases of phthisis pulmonalis (mostly of the ad-

vanced stage). In only one of these five cases was there any suspicion of middle ear tuberculosis.

The Path of Infection.—In the majority of cases of middle ear tuberculosis in children the infection reaches the ear through the eustachian tube. This may occur either by the infectious material passing through the lumen of the tube without involving its lining membrane or by the direct extension of the tuberculous disease from the nasopharynx along the lining membrane of the eustachian tube. There may be evidence of the existence of tuberculous disease in the adenoid vegetations of children affected with tubercle of the middle ear cleft. Infection of the temporal bone by the blood stream is probably rare in children. Henrici and others, however, hold that tubercular otitis is often due to a hematogenous infection. If it can be shown at operation that the tube and tympanum are healthy, and that the mastoid alone is diseased, the probability is that the infection has occurred by way of the blood stream.

Clinical Characteristics of Middle Ear Tuberculosis. (a) Mode of Origin.—This is, as a rule, of a quiescent character, and in 92 per cent no history of apparent pain or restlessness could be obtained either preceding or accompanying the onset of the ear affection. The mother's attention is first drawn to the ear by observing the presence of discharge. Enlarged periotic glands were noted in 95 per cent of the cases. In a number of these caseation and suppuration occurred.

The discharge from the ear is watery or flocculent—at least in the early stages before mixed infection occurs. Multiple perforations in the drumhead may be observed in adults, but in infants the sagging of the meatal will usually prevent inspection of the tympanic membrane. If mixed infection occurs the symptoms of an acute otitis media and mastoiditis may arise. Facial paralysis was present in 45 per cent of the cases observed by Turner.

Types (or Stages) of Tuberculous Otitis Media and Interna.—(1) Lupoid—in cases of lupus of the nose and throat. (2) An infiltrating form, which progresses rapidly and shows numerous tubercle bacilli but few giant cells. (3) A fungating or more chronic form with well marked tubercle follicles and few bacilli. (4) A necrotic form which tends to rapid caseation and destruction of the mucosa and bony capsule of

the labyrinth. The necrosis may be due to blocking of one or more of the arterial twigs supplying the labyrinth capsule by tuberculous endo- or peri-arteritis, but mixed infection is probably of even greater importance. (5) A fibro-ossifying type of tuberculous otitis interna may occur—i. e., a type in which nature makes considerable effort towards a spontaneous cure of tuberculous labyrinthitis by the formation of new fibrous tissue and bone. This type of tubercular disease is well recognized in the case of the long bones.

Pathologic Conditions Found at Operation.—Carious bone was found in 65 per cent and a sequestrum in 60 per cent. In some cases the outer antral wall lay loose and could be readily picked out with forceps. The granulations often presented a pale flabby appearance. Caseous material was noted in the antrum and tympanum in several cases. In nine instances the malleus and incus were absent.

Concomitant Affection of the Labyrinth.—In 35 of the series the mastoid operation was performed and the labyrinth was found to be necrosed in whole or in part in eight of these, or in 22 per cent. In addition, the external labyrinthine wall showed pathologic changes in 11 cases—i. e., 19 out of 35 cases showed labyrinthine changes (54 per cent). The lesions observed were superficial erosion and softening in the region of the external semicircular canal, facial canal and promontory.

In infants and very young children the ordinary tests of the labyrinth function can hardly be applied, but if the radical mastoid operation be performed it is possible, at the end of the operation, to apply cold lotion to the inner wall of the operation cavity, and the anesthetist is able to observe whether or not this procedure results in conjugate deviation of the eyes to the lower or nonoperated side.

Route of Infection from Middle Ear to Labyrinth.—Infection spreads to the labyrinth from the middle ear, as a rule, by way of the oval or round windows. The stapes footplate is soon eroded, but the endosteum lining the vestibule resists, and is pushed inwards in front of the mass of tuberculous granulation tissue before rupture occurs and the perilymph space of the vestibule is invaded. Invasion of the labyrinth through the secondary tympanic membrane is not resisted to the same extent. The prominence of the external semicircular

canal is occasionally the seat of a fistula into the labyrinth, but not nearly so often as in cases of cholesteatoma. On the other hand, the promontory is much more often diseased in tubercular than in simple purulent otitis media. Clinically, tubercular labyrinthitis, like tuberculous otitis media, appears to have a quiet onset.

Complications: Tuberculous pachymeningitis externa is frequently met with at operation. Hemorrhage from the internal carotid artery has been recorded. Tubercular thrombosis of the sigmoid sinus may occur. Tuberculous meningitis and tuberculous tumors are met with at postmortem examinations but they are usually regarded as parts of a general tuberculosis. Two of the patients who were operated upon died of tuberculous meningitis.

Treatment.—Brieger holds that the indications for treatment in tuberculosis of the middle ear are the same as those in the ordinary purulent forms of otitis media and mastoiditis. Ruttin has informed us that in Vienna they only operate on tuberculous otitis if the patient is suffering severe pain, as they consider it hopeless to get rid of all the disease. Probably the correct position lies between these two extremes. It is useless to perform a Schwartz operation. The radical operation is therefore indicated in all cases except those rare forms in which there is an apparently hemotagenous involvement of the mastoid process. At the radical operation it is advisable to expose freely the dura of the middle and posterior fossæ, because this membrane forms a much better barrier than the bone to the spread of tubercular disease. One can curette the eustachian tube from the mastoid wound, and remove tubercular adenoids if they be present. It is, therefore, not possible to get rid of the whole of the disease, so that one must depend to a great extent on the resisting powers of the patient, and for this reason it is very important that sanatoria should be provided for the after treatment of the surgical forms of tuberculosis. It is useless to send these cases of tuberculous otitis back to the slums from which so many of them come.

Pfannenstiel's method of treating tuberculosis of the nasal mucous membrane may be applied with advantage in the after treatment of tubercular otitis media.

Case 1.—W. Y., aged one year and three months; bottle fed; bilateral tuberculous otitis media; left facial paralysis one month before admission. Child has never had any pain in the ear. Foul pus in both meatus; shotty glands behind sternomastoid.

Operation on Left Ear: Antrum contained granulation tissue; bone around soft, and granulations in oval and round windows; malleus and incus absent. Later, operation on right ear. Two days after this second operation the child became very restless; rapid respiration; squinting; vomiting; bronchopneumonia present. Death.

Postmortem.—Tuberculosis of right lung and of bronchial glands; mesenteric glands enlarged and caseous; no tubercle of brain. (Guinea pigs inoculated from lymphatic glands removed from back of left ear showed definite tuberculosis. Granulation tissue from the ears showed small tubercular areas.)

Microscopic Examination of Right Ear (Case 1).—Eustachian tube: Tuberculous nodules present in submucosa. Tympanic cavity: Tuberculous granulation tissue is seen extending into the roof of the tympanum (Fig. 74) and the inner wall of the antrum, and can be traced from this for some distance through the nucleus of the labyrinth (fossa subarcuata). Oval window: The stapes is eroded and displaced towards the vestibule (Fig. 75). There is granulation tissue present between the footplate of the stapes and the endosteum of the vestibule. At the lower margin of the oval window the annular ligament is disintegrated, the endosteum is ruptured and the purulent infiltration has entered the perilymph space of the vestibule, and, penetrating Reissner's membrane, has entered the cochlear canal. Round window: The secondary tympanic membrane is greatly thickened, the niche of the round window being filled with swollen mucosa containing tubercular granulation tissue (Fig. 75). Labyrinth capsule: The lamellar bone over the promontory is eroded, but the cartilage bone is almost entirely healthy. The bony wall of the external canal prominence is also eroded (Fig. 76), but a fistula has not formed. Cochlea: There are curdled lymph and mononuclear cells in the scala tympani of the basal coil, especially just above

the round window membrane (Fig. 75). Vestibule: Just above and internal to the oval window the branch of the vestibular nerve to the utricle and cristæ of the external superior canals is infiltrated with pus cells.

Remarks.—The case was an early one of infiltration tuberculosis affecting the mucosa of the middle ear cleft. The infection had apparently spread up the tube and involved the mucosa of the tympanum, attic, and mastoid antrum; from the inner wall of the antrum it had spread along the vessels of the fossa subarcuata towards the cranium. Invasion of the labyrinth was just beginning.

Case 2.—J. N., male, aged nine months (a twin), had epileptic fits at the age of six weeks; a fortnight later the right ear began to discharge; the fits continued at intervals. At three months the right ear was operated upon at another hospital, but the discharge continued. Examination.—Child markedly wasted; mouth shows thrush; frequent vomiting. Child's head sweats profusely and is markedly retracted; photophobia present. Enlarged glands below the right ear, and behind the ear there is a fistula discharging pus. The right side of the face is paralyzed.

Operation: Large flat sequestrum removed from roof of middle ear. Sequestrum also removed from floor and another from the inner wall, opening up the cochlea, vestibule, semi-circular canals and fallopian aqueduct.

After operation progressive emaciation and glandular enlargement; wound cavity shows few granulations. Death two weeks after operation.

Postmortem: Tubercular nodule in right anterior corpus quadrigeminum and another in the occipital region. The dura of the right middle fossa showed tubercular thickening.

Microscopic Examination of Right Ear (Case 2).—Eustachian tube: The walls of the bony portion of the eustachian tube are destroyed, so that the dura (above) and the internal carotid artery (to the inner side and below) are infiltrated with tubercular tissue (Figs. 77 and 78). Tympanic cavity: The anterior part of the tympanum shows marked tubercular infiltration with caseation. The bony wall (cochlear capsule) is eroded—both lamellar and cartilage bone being involved

(Fig. 79). Oval and round windows: Absent through disease. The marrow spaces show tubercular granulation tissue, which extends up to the dura mater of the posterior fossa. Cochlea: All coils are filled with granulation tissue. No giant cell systems are seen, but there is a good deal of caseation. All membranous structures have disappeared. The outer wall of the cochlea is almost entirely eaten away. There is a fistula from the middle ear into all three scalæ of the cochlea (Fig. 79). (From the appearances present it is evident that there was a mixed infection of the labyrinth in this case.) Vestibule and Canals: There is a large fistula from the middle ear into the vestibule, and all membranous structures have disappeared except the aqueduct which, at the vestibular end, is plugged with granulation tissue and pus. The nucleus of the labyrinth has broken down into a tuberculous abscess, and the disease has spread along the fossa subarcuata (Fig. 80). The external canal has disappeared entirely, while the posterior canal contains pus in both ends. Internal Meatus: Shows greatly engorged and dilated vessels; there are a few pus cells in the fundus of the internal meatus.

Remarks.—This case was one of the severe type of tubercular otitis media and interna—the necrotic form. In spite of the large area of necrosis, which included the promontory, the external canal, part of the superior and posterior canals and almost the whole of the fallopian aqueduct, there is no evidence of gross disease of the labyrinthine artery in the internal meatus. The extensive necrosis seems to have been due to the mixed infection rather than to any gross disease of the artery supplying the labyrinth capsule.

Case 3.—H. C., male, aged nine months (breast fed). Nasal catarrh and obstruction since birth; left sided otorrhea without pain of ten weeks' duration; swelling behind left ear for eight weeks. Wilde's incision made by patient's doctor. Examination: Very marked adenoids; large kidney shaped perforation of left tympanic membrane.

Adenoid operation performed and later the radical mastoid operation. Much granulation tissue present. Head retraction developed; meningitic cry; internal strabismus; cerebrospinal fluid clear—slight increase in mononuclear cells. Death.

Postmortem: Tubercular meningitis and general tuberculosis.

Microscopic Examination of Left Ear (Case 3). Middle Ear.—The mucous membrane is greatly thickened and infiltrated with tubercle over the lower part of the promontory and in the niche of the round window. In the latter position giant cell systems are present. The stapes is absent and there is a large fistula into the labyrinth through the oval window (Fig. 83). There is a second fistula between the scala vestibuli of the basal coil of the cochlea and the tympanic cavity (Fig. 81). The round window membrane is greatly thickened and infiltrated. Cochlea: The basal coil of the cochlea in its lower part is entirely filled up with new connective tissue and bone; the new bone formation is especially marked in the scala tympani and occludes the opening of the perilymphatic duct. There is some new endosteal bone in the scala vestibuli. The spiral canal in the basal coil is filled up with small cells and the ganglion cells have disappeared. No trace of Corti's organ remains. In the middle coil there is formation of new connective tissue with a little new bone in the scala tympani. The cochlear canal itself is greatly dilated by contraction of the new connective tissue in the scala vestibuli, pulling on Reissner's membrane (Fig. 82). In the middle coil Corti's organ is represented by a low mound of cells in which pillar cells cannot be recognized. The modiolus is markedly infiltrated with small cells. Vestibule and canals: The vestibule shows a considerable amount of new fibrous tissue and bone formation from the endosteum (Figs. 83 and 84). There is a large cavity in the center, lined by tubercular granulation tissue which shows giant cells in parts and in others caseation and sequestrum formation. No membranous structures are visible. The canals are also entirely replaced by new formation of connective tissue and bone. Internal meatus: The nerves are infiltrated by small mononuclear cells. In parts the infiltration amounts to nodule formation (Fig. 81).

Remarks.—The well marked tubercle follicles present in the tympanic mucosa in this case point to a chronic form of tuberculosis. The condition of the labyrinth is interesting, as there is great evidence of an attempt on the part of nature to achieve a spontaneous cure of tubercular labyrinthitis by a process of

fibrosis and new bone formation. The invasion of the labyrinth appears to have taken place mainly through the oval window into the vestibule and, to a less extent, through the round window into the cochlea.

7. NEURITIS OF THE EIGHTH NERVE (MÉNIÈRE'S SYNDROME).

Ménière, in 1861, described the case of a young girl who, after a cold drive during her menstrual period, was attacked by sudden deafness, intense dizziness and vomiting. She died on the fifth day of her illness, and at the autopsy it was found that the semicircular canals and the vestibule contained hemorrhagic exudate. The cause of death was not discovered.

Although Ménière's classical case was described more than sixty years ago, there is still considerable confusion on the subject of Ménière's disease, and the term is frequently applied to any condition in which dizziness is associated with a lesion of the ear. It would probably be better to discard altogether the use of the words "Ménière's disease," but, if the term be retained, it should be kept for cases in which Ménière's symptoms appear in an apoplectiform manner.

These symptoms include "deafness, tinnitus, giddiness, nausea, vomiting, loss of balancing and nystagmus." This "disease picture" is that of a lesion of the membranous labyrinth or eighth cranial nerve. It should be associated with disease of the labyrinth, just as "tremor, rapid pulse and exophthalmos" are associated with the condition of hyperthyroidism.

Apart from injuries of the labyrinth—e. g., fracture of the cranial base, war injuries, etc.—most of the cases of Ménière's symptom complex are probably due to neuritis of the eighth nerve, associated with toxemia or exposure to cold. Except in the bleeding diseases—e. g., leukemia and pernicious anemia, etc.—hemorrhage into the labyrinth has never been proved by microscopic examination.

As syphilis is a frequent cause of Ménière's syndrome, the Wassermann reaction of the blood and cerebrospinal fluid should always be tested. Tabetic auditory nerve deafness is one of the less common complications of locomotor ataxia and may be due to a degenerative neuritis in the nerve trunk or an affection of the auditory nucleus. Lehmann has found that nerve deafness of toxic origin may occur in enteric fever: the vestibular apparatus is not affected, while permanent injury to

the cochlear nerve is rare. Recently recurring attacks of Ménière's symptoms have been attributed to toxic neuritis or neurolabyrinthitis due to absorption of toxin from a focus of infection—e. g., pyorrhea alveolaris, septic tonsils, appendicitis or to gastrointestinal disturbances in people whose auditory or vestibular apparatus is specially sensitive. Ruttin has pointed out that as the lymphatic spaces of the inner ear are continuous it is almost impossible to conceive of an isolated affection of the cochlear or vestibular apparatus arising within the labyrinth itself. On the other hand, the cochlear and vestibular nerves may be affected separately by toxins which have a special affinity for one or the other division. **When the cochlear apparatus alone is affected we have tinnitus and nerve deafness. When the vestibular apparatus alone is involved dizziness is the chief symptom. The complete Ménière syndrome is present only when the labyrinth or both parts of the eighth nerve are affected.** In chronic nephritis labyrinthine deafness is due to a toxic rather than an inflammatory, circulatory or edematous change. It is usually associated with a very considerable amount of vertigo. Degenerative neuritis occurs in cases of diabetes mellitus—the cells of the spiral ganglion being first affected. A similar neuritis has been described in gout and in cancerous cachexia. Toxic paralysis may be caused by quinin, salicin or salicylates, more rarely by alcohol, tobacco or lead poisoning. In many instances, however, the etiology of the neuritis is obscure.

A Case Presenting Ménière's Symptoms Along with Facial Paralysis.—A. McR., male, piermaster, aged 65, had never suffered from deafness up to the time of his last illness. On January 12, 1907, he had a long day of exposure to cold on the pier. The following morning he woke up suffering from extreme dizziness, deafness and noises in the left ear. The left side of his face was paralyzed. He also complained of pain in the head and limbs and did not leave his bed for a week. He felt as if surrounding objects were moving around him. There was no herpes auricularis. When he got up he had to hold on to furniture in order to get about the house. Examination November 2, 1907: Tympanic membrane normal, watch heard by right ear at two inches; by the left it is not heard even on contact. Forced whisper heard by right ear at

two feet and by left ear at one foot. All tuning forks heard by air conduction by left ear. Rinne's test positive on both sides. Patient can stand with his feet together and eyes shut without swaying. Left facial paralysis present, but patient can taste salt on the anterior part of the tongue on the left side. No response to faradic current on the left side of the face; the affected muscles contract slowly to galvanism, but there is no alteration of the polar reaction. The arterial walls are thickened. The lungs both reveal extensive chronic tuberculosis. Joints of hands swollen and painful. The diagnosis made by the late Dr. Alexander Bruce was that of neuritis of facial and auditory nerves in the internal auditory meatus. "The absence of evidence of middle ear disease and the retention of the sense of taste, and the associated deafness, dizziness and facial paralysis indicated that the lesion was probably situated between the side of the pons and the bottom of the internal auditory meatus."

The patient died suddenly February 5, 1908.

Postmortem: Atrophy of second, third, fourth and sixth cranial nerves on left side; extensive chronic pulmonary tuberculosis; subacute interstitial nephritis; milky thickening of arachnoid over inferior surface of cerebellum and circle of Willis.

Examination of left inner ear and auditory and facial nerves: The inner ear was almost normal; there was no hemorrhage in the endo- or peri-lymphatic spaces of the cochlea, vestibule or canals. The membranous labyrinth was also normal, as was the spiral ganglion. The neuro-epithelium of the utricle, sacule and of the cristæ ampullarum was well preserved. In the internal meatus there was a considerable amount of small cell infiltration (Fig. 85), between the epineurium and the seventh and eighth nerves; this was most marked above the seventh nerve. The vessels in the internal auditory meatus were markedly thickened, and the muscular coat had undergone a hyalin change. The infiltration of white cells did not extend into the Fallopian canal and did not therefore affect the geniculate ganglion.

8. TUMORS OF THE EIGHTH NERVE.

In the vast majority of if not in all cases the growth starts in the internal auditory meatus and only later invades the

cranial cavity. Cushing points out that owing to the characteristic chronology of their symptoms, acoustic tumors may, as a rule, be sharply distinguished from all others. The writer hopes that in the future otologists may be able to diagnose these cases in the early or otologic stage, when operative treatment holds out a much better chance of recovery than exists at the later periods when, according to Cushing, all we can effect is an extensive decompression operation combined with removal of "as much as possible" of the growth.

Etiology: (1) Heredity.—Neurofibroma of the eighth nerve is related to general neurofibromatosis, which may occur as a family disease. Feiling and Ward have published a paper on a "familial form of acoustic tumor."

(2) Age.—Henschen has collected 125 cases; second decade, 9; third, 20; fourth, 26; fifth, 27; sixth, 6; seventh, 1. The symptoms of an acoustic tumor rarely occur before the third decade.

(3) Infection.—Hessler has collected nineteen examples of acoustic tumor in patients who had suffered from middle ear disease. Siebenmann and Nager, on the other hand, believe that these acoustic tumors are associated with intrauterine meningitis.

Pathology.—Virchow has classified neuromata into (1) true and (2) false. Some say that true neuromata do not exist. False neuromata are composed of connective tissue derived from the nerve sheath and include acoustic neurofibromata. The endoneural connective tissue between the fibers is the seat of the pathologic process. The perineurium at first remains unchanged and forms the capsule of the tumor. Henschen states that five cases of "solitary" but symmetrical tumors of both eighth nerves have been recorded, and further, that the cases of multiple neurofibromatosis with acoustic tumor number sixteen.

The tumors vary in size from a hazelnut to a small apple. They have an uneven nodular surface and a grayish pink color. On section the tumors may show cysts or spaces filled with blood.

Histology.—Of 107 cases collected by Henschen, 86 showed a connective tissue structure and 21 appeared to be gliomatous. The latter appearance may be due to edema. Greenfield

states that the tumor is composed of cells of the connective tissue type. These cells have an elongated nucleus and the cytoplasm tails off into a spindle shaped process at either end. There are areas of looser texture, suggesting myxomatous or glial tissue. They are probably mixed tumors, gliofibromas composed of germinal tissue—i. e., they are tissue "rests." They probably spring from the crest of nerve tissue lying between the ectoderm and the dorsal surface of the neural tube from which the seventh, eighth, ninth and tenth nerves arise. Henschen believes that the tumors originate in the inferior vestibular branch of the distal portion of the nerve in the internal meatus. He points out that between the branches of the eighth nerve in the internal meatus there is present embryonic connective tissue and that this explains why the eighth nerve is so often the seat of tumor. Orzechowski, on the other hand, thinks that the tumors are gliomatous and have little or nothing to do with the acoustic nerve and holds that they proceed from a malformation of the median wall of the lateral recess. Bruce and Dawson came to the conclusion that fusiform nucleated cells, linked on to one another, form embryonic nerve fibers—i. e., that nerve fibers are formed by the same process which gives rise to neuromata in the repair of divided nerves. A neuroma thus arises from the perverted function of the neurilemma sheath.

The eighth nerve consists of two parts: (a) A central non-medullated, and (b) a peripheral medullated portion. The junction of these two parts occurs just within the internal auditory meatus at its cranial end. Henschen states that in all cases of tumor only the distal portion of the nerve was affected, just beyond the junction of the two parts. In the case of large tumors there is always a cone shaped portion of the growth in the internal meatus.

Changes in the Labyrinth.—Alexander and Fränkl-Hochwart have examined the labyrinth in one case of acoustic neuroma and found degenerative atrophy of the cochlear nerve and spiral ganglion; atrophy of Corti's organ and the stria vascularis; dropsical degeneration of the spiral ligament. Alexander in another case found the round window membrane thickened by a layer of connective tissue on its cochlear side. The perilymph spaces contained a network of fibrin threads (Alex-

ander thinks that this fibrin network becomes organized into connective tissue); the bony spiral lamina filled with connective tissue; all the labyrinth nerve endings were degenerated. Corti's organ was absent in parts. Frankenstein in a case of multiple neurofibromatosis found the hollow spaces of the modiolus filled with tumor. The scala tympani of the lower coil was also filled with tumor; above this there was exudate. The nerve canals and vestibule were filled with tumor.

The microscopic examination of the labyrinthine nerve endings in cases of tumor of the nervus octavus is of interest in connection with the question of secondary degeneration of sensory nerve endings after they have been cut off from their ganglia. Wittmaack has cut the seventh and eighth nerves in animals before entrance into the internal meatus and has produced degenerative changes in the cochlear nerve ending in the labyrinth. These results are in conflict with the hitherto accepted idea regarding secondary degenerations. Waller's law states that after supraganglionic division of a sensory nerve there is no degeneration of the peripheral neurone. In cases of compression of the eighth nerve by neurofibroma Panse, and also Alexander, found atrophy of the spiral ganglion and loss of the sensory cells of Corti's organ. These changes may, however, have been due to compression of the internal auditory artery.

Case I.—M. McQ., female, aged 75, was admitted to a poor-house hospital on April 30, 1909, complaining of dizziness, deafness and noises in the left ear, of five years' duration. She could not walk without assistance. Rombergism marked. No note as to nystagmus. Patient has well marked arteriosclerosis.

Functional examination: C_{32} and C_{64} not heard by either ear. C_{128} , C_{256} , C_{512} and C_{1024} are all heard faintly by the right ear (side of tumor) but not by the left. C_{2048} not heard by either ear. Schwabach shortened. Galton's whistle not heard at all. Vestibular apparatus not tested. December 15, 1909: Patient can walk without help; deafness as before. Vision very bad. October, 1910: Dizziness worse; patient fell. January, 1911: Patient better. June, 1911: Indefinite headache. January, 1912: Patient completely deaf and bedridden. October, 1912: Sudden paralysis of right arm, leg and face. Death. Postmortem showed a tumor the size of a cherry involving the

eighth nerve on the right side. Gelatinous fluid present in air cells of mastoid on both sides.

Examination of right ear (side of acoustic tumor): Mucosa of tympanic cavity and antrum slightly thickened. Delicate connective (?) tissue, very like tumor tissue, is seen in the scala tympani of the cochlea (Figs. 86, 87 and 88). There are large vessels in this tissue. The modiolus and spiral canal are infiltrated with the new connective or neuroglia (?) tissue (Figs. 86 and 87). In the lower part of the basal coil Corti's organ has quite disappeared (Fig. 87). In the upper part, Corti's organ is recognizable, and there are some ganglion cells left (Fig. 86). There is new bone formation in the scala tympani of the middle coil and in the basal coil (Figs. 86 and 87). The neuroepithelium of the utricle and saccule is well formed and the otolith membrane is present. The crista of the superior canal is atrophied when compared with the well formed cristæ of the lateral and posterior canals. New connective tissue formation is seen in the perilymph space of all the canals toward the convexity. The internal meatus is dilated and its posterior margin eroded by tumor tissue (Fig. 88). The tumor did not reach the fundus. The tumor, which shows large blood spaces, is composed of gliomatous or myxomatous tissue.

Examination of the left ear: The basal coil of the cochlea contains a considerable quantity of new bone and connective tissue (?) formation (Fig. 89). The scala vestibuli also shows new connective tissue (?) and bone formation. The scala media is greatly dilated. Corti's organ is present in all coils, and there is only slight atrophy of the spiral ganglion cells. Canals as on right side. The cochlear nerve, as is usual in old people, shows well marked corpora amylacia but is otherwise normal. The vestibular nerve is very atrophic.

The interesting thing in this case is the presence on both sides of changes which are usually associated with the presence of an old labyrinthitis. There is, however, very little evidence of middle ear suppuration. It is possible that the labyrinth has been infected from the meninges, as has been suggested by Siebenmann and Nager. Lastly, the presence of a tumor of the eighth nerve on the right side suggests that the condition of the inner ears may be due to an end stage of "choked

labyrinth." When the writer saw the patient in 1909 (three years before death) she could hear a little with the right ear (side of the tumor) but not at all with the left ear. This fact does not support the view that the changes in the two labyrinths were the result of the tumor in the right eighth nerve, although, on other grounds, this appears to be the most probable explanation.

Case 2.—A. T., female, aged 25, was an inmate of a hospital for incurables. Clinical details of the case are almost entirely wanting. The patient's face was very expressionless, and she was mentally dull and bedridden. She was blind and had double optic neuritis.

Postmortem examination: Outer (cranial) surface of dura, over almost its entire extent, was covered with small pedunculated tumors which varied in size from a hayseed to a split pea. On removal of the dura the inner surface of the cranial bones appeared as if deeply pitted by smallpox. In the region of the right internal auditory meatus and angle between the pons and cerebellum there was a tumor mass about the size of a walnut.

Examination of right ear: The tumor is eroding the posterior surface of the petrous bone (Figs. 90 and 91). The spiral ganglion in the lower part of the basal coil is infiltrated with tumor tissue and the ganglion cells have disappeared. The scala tympani is occupied by tissue resembling that of the tumor (Figs. 91 and 92). Internal to the round window membrane there is some new bone formation (Fig. 92). The perilymphatic aqueduct is filled with tumor. Corti's organ is unrecognizable in the lower part of the basal coil, although the patient was only 25 years old. In the middle and apical coils Corti's organ is fairly well formed. The neuroepithelium of the saccule and utricle is normal. The cristæ of the canals are practically normal. The internal meatus is greatly dilated by the tumor, especially at its inner end (Fig. 91). No giant cells are present, although the bone has been eroded. There is no appearance in this case of any otitis media. The condition of the dura mater and of the cerebral surface of the cranial bones was very interesting. The outer surface of the dura showed numerous small and rather pedunculated swellings which had eroded the inner or cranial surface of the

bones which form the "brain case" (Fig. 93). Cushing holds that these small swellings on the dura are hypertrophied arachnoid villi or Pacchionian bodies. If Cushing is correct in this view, one would expect the swellings to be present in other cases with increased intracranial pressure—e. g., in other varieties of intracranial tumor. Professor Shennan, however, has informed the writer that such is not the case. Professors Lorrain Smith and Theodore Shennan, Dr. J. W. Dawson and Mr. Richard Muir have kindly examined the sections of these projections from the dura and have given their opinions that the tumors are composed of nerve tissue similar to that seen in gliomata. Key holds that the protrusions are small gliomatous "metastases" in the arachnoid. Without adopting the idea of metastasis or secondary deposits of glioma due to blood infection, it may well be that the small swellings in the brain membranes are due to overgrowth of glia tissue—in other words, that they are caused by a diffuse gliomatosis.

Case 3.—W. H., male, aged 43, had been deaf in the right ear for six years but was otherwise quite well till two years ago, when he was operated on for appendicitis. Soon after he began to complain of dizziness and unsteady gait and of occasional vomiting. Patient stated that he tended to stagger to the right side. He also noticed weakness of the right side when fatigued and right sided headache. A little later diplopia was noted. Examination by Professor Bramwell showed nystagmus on looking to the right and left, marked deafness in the right ear. The grasp of the right hand was 85, while that of the left was 120. (The patient was a right handed man.) The finger-nose test was badly performed on the right side. Ophthalmoscopic examination showed distinct optic neuritis. The right knee jerk was more active than the left. Professor Bramwell was of opinion that the hemiplegia did not present any pyramidal characteristics and was probably due to disturbance of the cerebellum on the right side. He thought that the patient's condition was probably caused by an extracerebellar growth originating in the eighth nerve. Later, a change in the position of the head was noted. It was held slightly depressed to the left, with the chin pointing to the right—the position assumed by the head of a monkey after one lateral lobe of the cerebellum has been removed. This posi-

tion could not be regarded as of localizing value, since the head is sometimes depressed towards the side of the tumor and sometimes toward the opposite side.

Otologic Examination: Cochlear apparatus: The loud voice was not heard by the right ear when the noise box was placed in the left ear. Weber lateralized to the left (good) ear. Rinne "infinite negative" on right side. Vestibular apparatus: Marked nystagmus to the right, less marked to the left; there was also distinct nystagmus to the left on looking up. Pointing test normal. No Rombergism. On cold syringing of the right ear there was no increase in the nystagmus to the left. Galvanic reaction: With the positive pole applied to the right ear it required 14 ma. to make the patient fall to the right. The same, however, prevailed on the left side. There was no facial paralysis and the patient could appreciate salt on the anterior part of the tongue on both sides.

X-ray examination: This appeared to show that the internal auditory meatus was larger on the affected than on the normal side.

Operation by a general surgeon. The tumor, however, was not reached, even with the finger.

Postmortem: Tumor the size of a large walnut in the cerebellopontine angle on the right side.

Examination of right ear: The growth has eroded the bone above and in front of the cochlea (Figs. 94 and 97). The lamellar bone has yielded much more easily to erosion than the cartilage bone. Tumor tissue intervenes between the internal carotid artery and the cochlear capsule (Fig. 97). The new growth indeed comes right up to the carotid canal. The tumor appears rather to have compressed the cochlear nerve than to have infiltrated it (Fig. 95). The basal coil of the cochlea shows both scala tympani and scala vestibuli full of fibrinous exudate (Figs. 95 and 96). Reissner's membrane is bulged upwards. Corti's organ is not well formed in the lower part of the basal coil. The cochlear ganglion cells appear to be shrunken. The utricle and saccule are dilated (Fig. 94) but the neuroepithelium is normal. In the perilymph space of the vestibule, between the saccule and the oval window, granular fibrinous material is present (Fig. 94). The canals show exudate in both endo- and peri-lymph spaces, but

the cristæ and cupulæ are normal. The internal meatus is enormously enlarged by the tumor (Figs. 94 and 97). The whole anterior wall of the internal meatus has been eroded and the dural lining is present only in the fundus. The tumor tissue invades the saccus endolymphaticus. This may account for the dilated condition of the utricle and saccule.

Remarks.—In this case the condition of the labyrinth differed from that seen in Cases 1 and 2. There was no new connective tissue or bone formation. Although deafness in the affected ear had been present for six years, it is possible that Case 3 was of comparatively short duration when contrasted with Cases 1 and 2. If this is so, we may look on the fibrinous exudate as the early stage of "choked labyrinth," while the formation of new connective tissue and bone represents the late stage. In all three cases there was definite evidence of degeneration of the nervous structures of the cochlea, whereas the vestibular end organs were comparatively well formed.

Fig. 1.—Coronal section No. 21. 1, Tensor tympani; 2, eustachian tube; 3, floor of middle cranial fossa; 4, eighth nerve in the internal meatus; 5, posterior surface of petrous bone; 6, glossopharyngeal nerve and opening of aqueduct of cochlea; 7, marrow spaces.

Fig. 2.—Coronal section No. 97. 1, Tensor tympani; 2, apical coil of cochlea; 3, middle coil; 4, cochlear nerve in internal meatus; 5, cranial end of aqueduct of cochlea (perilymph); 6, basal coil of cochlea; 7, carotid canal; 8, tubal portion of tympanic cavity.

Fig. 3.—Coronal section No. 127. 1, Tensor tympani; 2, helicotrema; 3, canal for great superficial petrosal nerve; 4, modiolus; 5, facial nerve; 6, cochlear aqueduct; 7, edge of cartilage bone capsule of cochlea; 8, carotid canal; 9, tubal cells; 10, tubal part of tympanic cavity.

Fig. 4.—Coronal section No. 175. 1, Tensor tympani; 2, facial nerve passing above cochlea; 3, vestibular ganglion; 4, cochlear aqueduct and vein accompanying it; 5, jugular bulb; 6, air cells in floor of tympanic cavity; 7, tympanic cavity.



Fig. 1.

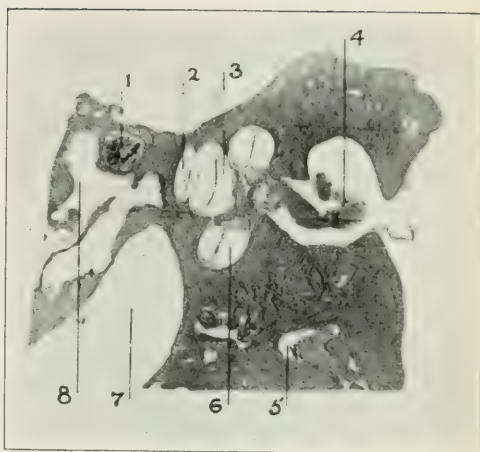


Fig. 2.

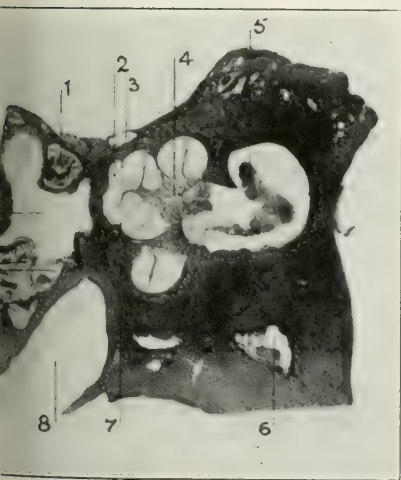


Fig. 3.

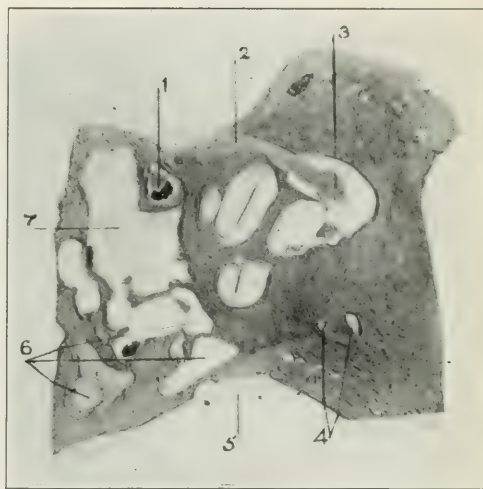


Fig. 4.

Fig. 5.—Coronal section No. 244. 1, Anterior part of epitympanic cavity; 2, facial nerve; 3, anterior part of utricle; 4, cochlear opening of perilymphatic aqueduct; 5, jugular bulb; 6, tympanic air cells; 7, tympanic membrane; 8, external meatus; 9, tympanic cavity; 10, sacculus; 11, tensor tympani.

Fig. 6.—Coronal section No. 295. 1, Tensor tympani; 2, facial nerve; 3, utricle; 4, ampulla of superior canal with crista; 5, opening of crus commune; 6, superior canal; 7, sinus of posterior canal with crista quarta; 8, niche of round window; 9, jugular bulb; 10, promontory; 11, external meatus; 12, handle of malleus attached to drumhead; 13, head, and 14, footplate of stapes.

Fig. 7.—Coronal section No. 320. 1, External attic; 2, head of malleus; 3, facial nerve; 4, two ends of lateral canal; 5, superior canal; 6, fossa subarcuata; 7, aqueduct of vestibule; 8, two ends of posterior vertical canal; 9, jugular bulb; 10, tympanic cavity; 11, external meatus; 12, tympanic membrane; 13, stapes; 14, Prussac's space.

Fig. 8.—Coronal section No. 385. 1, Head of malleus; 2, body of incus; 3, external canal; 4, air cells; 5, endolymphatic sac; 6, two ends of posterior canal; 7, jugular bulb; 8, drumhead; 9, sinus tympani.



Fig. 5.



Fig. 6.

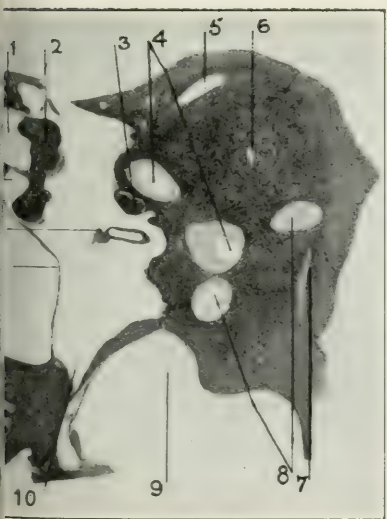


Fig. 7.

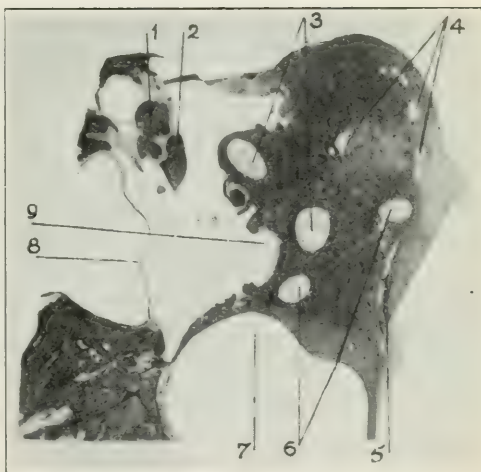


Fig. 8.

Fig. 9.—Coronal section No. 450. 1, Posterior part of drumhead; 2, external acoustic meatus; 3, outer wall of aditus; 4, short process of incus; 5, roof of tympanic antrum; 6, prominence of lateral canal on inner wall of aditus; 7, perilymph space of lateral canal; 8, air cells on inner wall of tympanic antrum; 9, two ends of posterior vertical canal; 10, saccus endolymphaticus; 11, air cells below labyrinth; 12, sinus tympani; 13, stapedius muscle; 14, facial nerve.

Fig. 10.—Coronal section No. 523. 1, Roof of mastoid antrum; 2, cavity of antrum; 3, posterior canal; 4, air cells; 5, saccus endolymphaticus; 6, air cells; 7, sinus tympani; 8, facial nerve; 9, chorda tympani; 10, external meatus; 11, border air cells overhanging external meatus.

Fig. 11.—Horizontal section No. 140. 1, Outer wall of attic; 2, body of incus; 3, head of malleus; 4, anterior ligament (remains of Meckel's cartilage); 5, inner attic; 6, geniculate ganglion; 7, basal coil of cochlea; 8, ampullary and smooth ends of superior vertical canal; 9, fossa subarcuata; 11, prominence of lateral canal.

Fig. 12.—Horizontal section No. 155. 1, Outer wall of attic; 2, malleus; 3, tendon of tensor tympani; 4, facial nerve; 5, middle coil of cochlea; 6, basal coil; 7, internal meatus with facial nerve to right and vestibular nerve to left; 8, ampulla of lateral canal, with crista; the short line points to the superior vertical canal; 9, fossa subarcuata; 10, tympanic antrum.

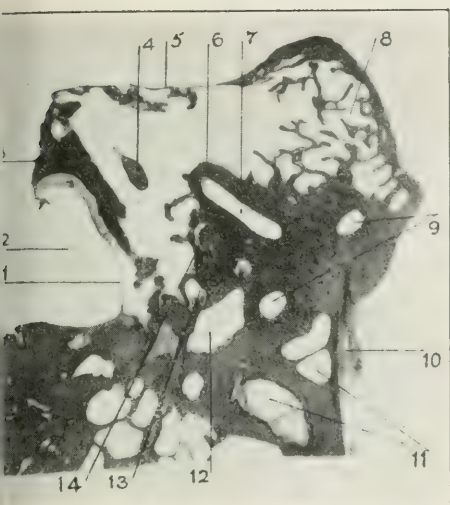


Fig. 9.

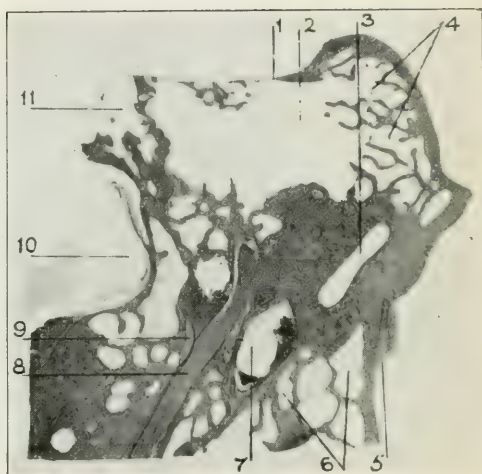


Fig. 10.

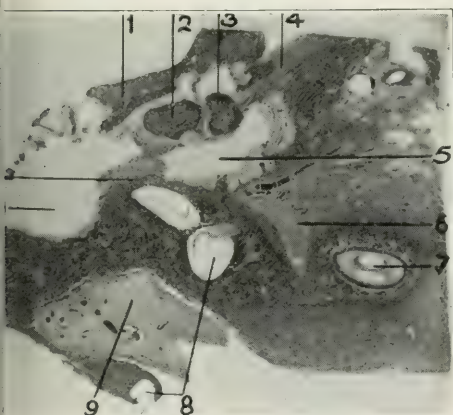


Fig. 11.

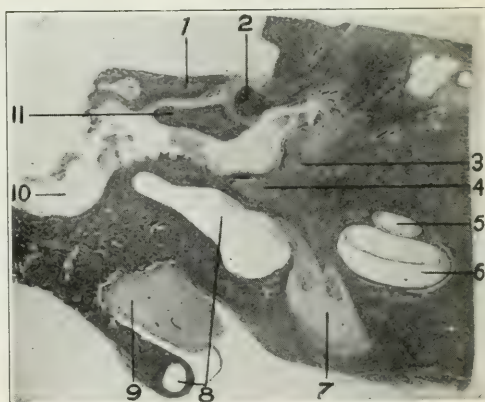


Fig. 12.

Fig. 13.—Horizontal section No. 205. 1, Footplate of stapes; 2, posterior part of annulus tympanicus; 3, handle of malleus; 4, tympanic cavity containing exudate usually found in the newborn child; 5, helicotrema of cochlea; 6, anterior wall of internal auditory meatus; 7, internal meatus with nerves; 8, saccule; 9, utricle; 10, crus commune; 11, lateral canal.

Fig. 14.—Horizontal section No. 255. 1, Facial nerve; 2, long process of incus; 3, head of stapes; 4, handle of malleus; 5, eustachian tube; 6, basal coil of cochlea; 7, internal meatus; 8, saccule; 9, footplate of stapes; 10, junction of crus commune with utricle; 11, smooth end of lateral canal opening into vestibule; 12, posterior canal.

Fig. 15.—Horizontal section No. 320. 1, Facial nerve; 2, handle of malleus with drumhead; 3, anterior part of annulus tympanicus; 4, eustachian tube; 5, carotid artery with accompanying veins and nerves; 6, basal coil of cochlea; 7, internal meatus; 8, junction between perilymph space of vestibule and scala vestibuli of cochlea; 9, vestibule; 10, lower part of utricle; 11, groove for saccus endolymphaticus; 12, posterior vertical canal; 13, sinus tympani; 14, stapedius muscle.

Fig. 16.—Horizontal section No. 350. 1, Facial nerve; 2, stapedius; 3, niche of round window; 4, drumhead below level of malleus; 5, scala vestibuli of basal coil; 6, tympanic opening of eustachian tube; 7, carotid canal; 8, scala tympani; 9, membrane of round window; 10 and 11, two ends of posterior vertical canal.

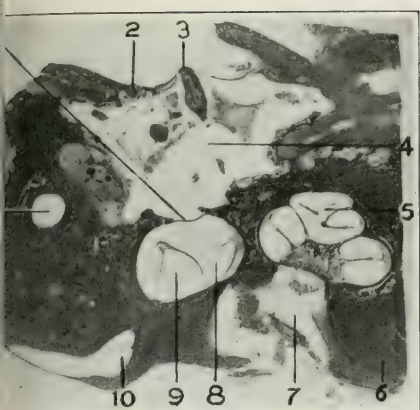


Fig. 13.

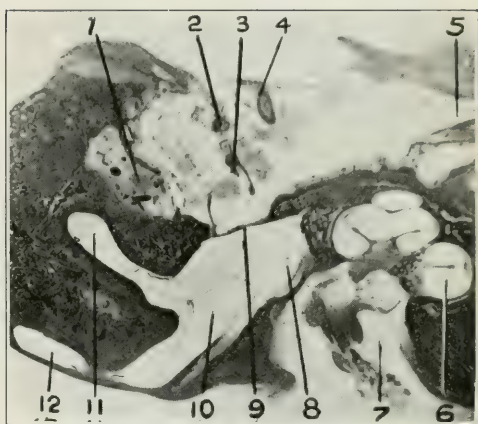


Fig. 14.

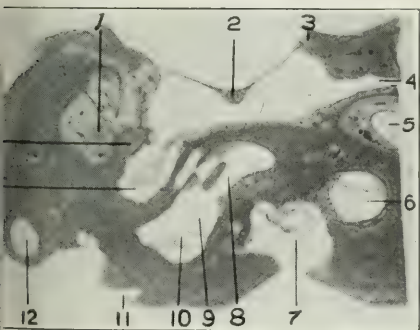


Fig. 15.

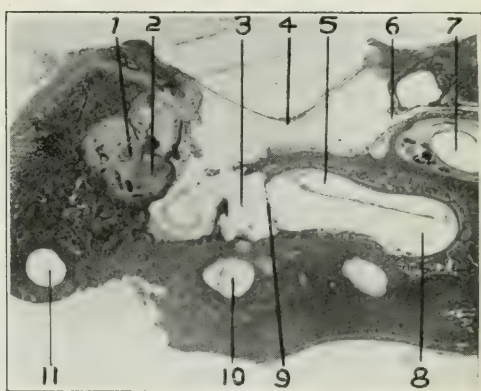


Fig. 16.

Fig. 17.—Case 1. Fracture of cranial base without fracture of labyrinth capsule. J. P., male, aged 54. Horizontal section through right ear, No. 122. 1, Footplate of stapes; 2, malleus; 3, hemorrhage; 4, incus; 5, vestibule; 6, posterior canal; 7, region of saccus endolymphaticus; 8, internal meatus; 9, basal coil of cochlea.

Fig. 18.—Case 1. Section through tympanic cavity. 1, Hemorrhage posterior to malleus and incus; 2 and 3, malleus; 4, incus; 5, stapes; 6, hemorrhage around facial canal; 7, facial nerve.

Fig. 19.—Case 1. Horizontal section through facial nerve and upper part of internal meatus. 1, Facial nerve; 2, geniculate ganglion; 3, basal coil of cochlea; 4, facial nerve showing ganglion cells along its course; 5, vestibular nerve with ganglion cells; 6, upper part of vestibule; 7, hemorrhage along branch of vestibular nerve to crista of lateral canal.

Fig. 20.—Case 1. Horizontal section No. 170. 1, Hemorrhage in modiolus; 2, blood in perilymphatic aqueduct; 3, cochlear opening of the aqueduct.

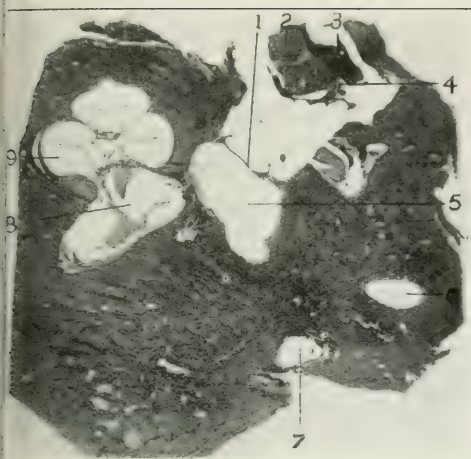


Fig. 17.



Fig. 18.

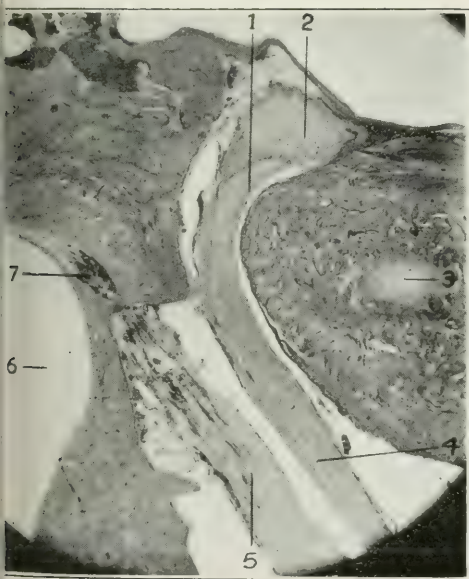


Fig. 19.

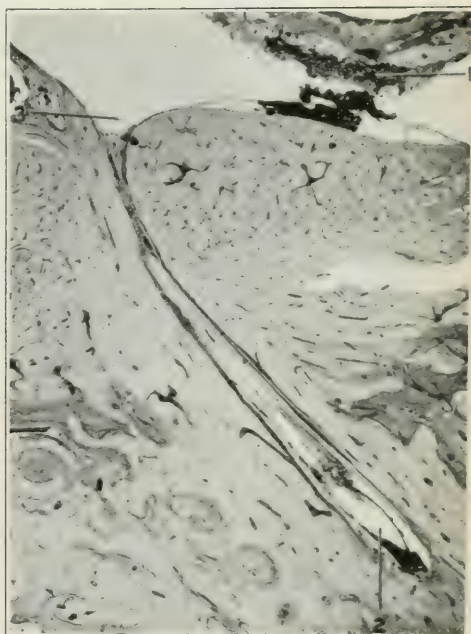


Fig. 20.

Fig. 21.—Case 2. Horizontal section through right ear. 1, Line of fracture on inner wall of attic—below this in the photograph there is blood in both peri- and endolymphatic spaces of the superior and lateral canals; 2, hemorrhage in endolymph space of lateral canal; 3, blood in air cell on inner wall of mastoid antrum; 4, line of fracture reaches a very large air cell, internal to the labyrinth; the line from 4 crosses the fossa subarcuata; 5, hemorrhages in the upper part of the basal coil of the cochlea.

Fig. 22.—Case 2. Horizontal section through right ear in the region of the oval window. 1, Fracture passes through footplate of the stapes; 2, lateral canal with blood in endo- and perilymph spaces; 3, hemorrhage in air cell internal to the labyrinth; 4, smooth end of superior canal free from blood; 5, fracture through vestibule from 1 and reaches 5; 6, hemorrhage in the vestibular nerve; 7, cochlear nerve with slight bleeding at the base of the modiolus; 8, basal coil of cochlea with blood in scala tympani and vestibule.

Fig. 23.—Case 2. Horizontal section through right ear in region of round window. 1, Line of fracture; 2, blood in convexity of posterior canal; 3, endolymphatic aqueduct free from hemorrhage; 4, ampulla of posterior canal with blood in it; 5, inner end of fracture reaches internal auditory meatus; 6, scala tympani of basal coil with hemorrhage; 7, cochlear canal contains blood.

Fig. 24.—Case 3. Vertical section. 1, Tensor tympani; 2, scala tympani of upper part of basal coil, filled with hemorrhagic exudate; 3, upper end of fracture; 4, the cochlear nerve surrounded by meningitis; 5, aqueduct of cochlea; 6 lower end of fracture; 7, basal coil filled with exudate; 8, exudate in tubal portion of tympanic cavity.

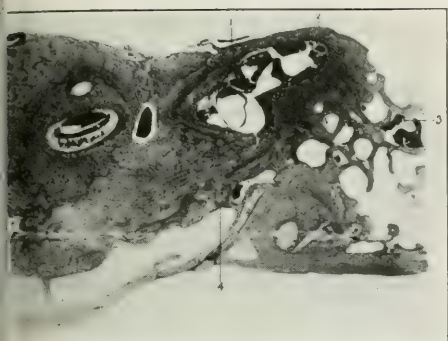


Fig. 21.

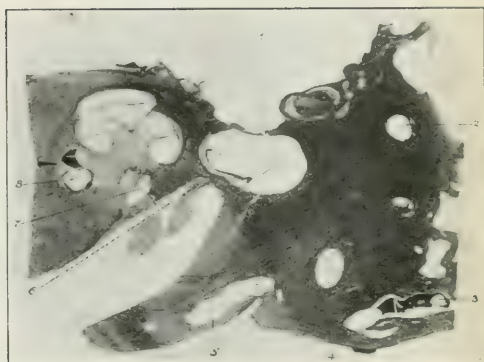


Fig. 22.

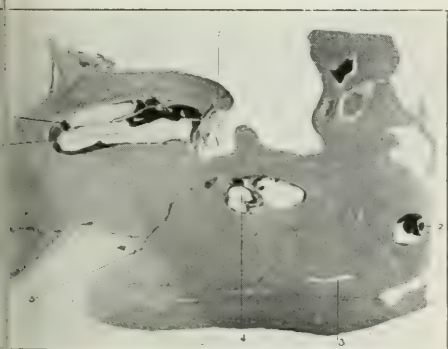


Fig. 23.

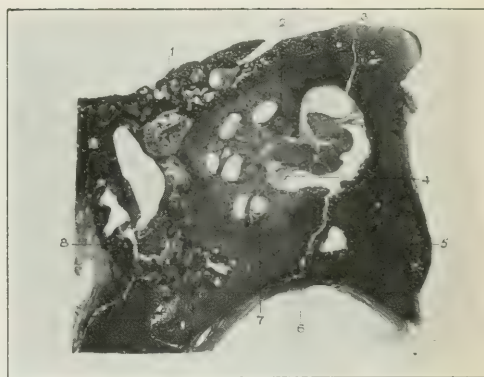


Fig. 24.

Fig. 25.—Case 3. Vertical section. 1, Facial nerve; 2, upper end of fracture, which passes into vestibule; 3, hemorrhagic purulent exudate in vestibule; 4, fracture through bony spiral lamina; 5, exudate in scala tympani; 6, lower end of fracture; 7, hemorrhagic exudate in tympanic cavity; 8, perforation of tympanic membrane.

Fig. 26.—Case 3. Vertical section. 1, Head of malleus; 2, fracture of tympanic roof; 3, superior vertical canal; 4, two ends of lateral canal; 5, ductus endolymphaticus; 6, two ends of posterior vertical canal; 7, exudate in tympanic cavity; 8 and 10, fracture of external meatus; 9, tympanic membrane below perforation.

Fig. 27.—Case 3. Vertical section. 1, Head of malleus; 2, exudate in attic; 3, two ends of lateral canal; 4, superior canal; 5, exudate in air cell internal to labyrinth; 6, two ends of posterior canal; 7, exudate in sinus tympani; 8, sinus tympani; 9, fracture of anterior wall of external meatus; 10, external meatus; 11, perforation of drumhead; 12, long process of incus.

Fig. 28.—Case 4. Vertical section. 1, Superior canal; 2, position which should be occupied by incus; 3 head of malleus; 4, exudate in tympanum; 5, stapedius muscle; 6, two ends of posterior canal; 7, two ends of lateral canal.

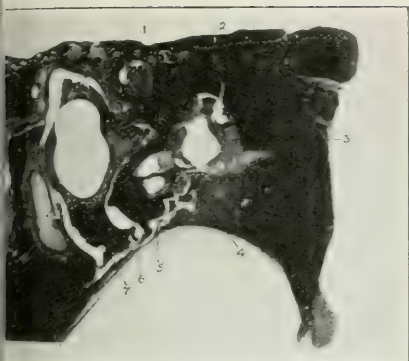


Fig. 25.



Fig. 26.



Fig. 27.



Fig. 28.

Fig. 29.—Case 4. Vertical section. 1, Connective tissue in gap left by old fracture; 2, long process of incus; 3, articular surface of incus; 4, tip of short process of incus which has remained attached to floor of aditus while the rest of the bone became dislocated upwards and backwards into the antrum; 5, epidermic lining of external meatus peeling off; 6, exudate in air cells behind labyrinth; 7, air cells with inflamed lining membrane on inner wall of antrum.

Fig. 30.—War Injury. Case 1. Direct injury to the ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, etc. Horizontal section through attic of left ear. 1, Malleus dislocated off incus; 2, facial nerve; 3, ampulla of superior canal; 4, perilymph space of lateral canal; 5, body of incus—note the thickened, engorged and infiltrated mucosa of the attic surrounding the ossicles.

Fig. 31.—Case 1. Section through tympanic cavity, showing: 1, perforation of drumhead; 2, greatly thickened tympanic membrane; 3, handle of malleus separated from the drumhead; 4, long process of incus, which is eroded; 5, fracture of stapes; 6, facial canal—the nerve is absent (artefact); 7, corda tympani nerve; 8, posterior part of drumhead.

Fig. 32.—Case 1. Horizontal section through region of oval window, showing 1, thickened and infiltrated drumhead; 2 handle of malleus separated from drumhead; 3, part of tympanic membrane carried inward with drumhead; 4, basal coil of cochlea with hemorrhage; 5, posterior part of annular ligament, infiltrated; 6, joint between incus and stapes—note the pus in the tympanic cavity.

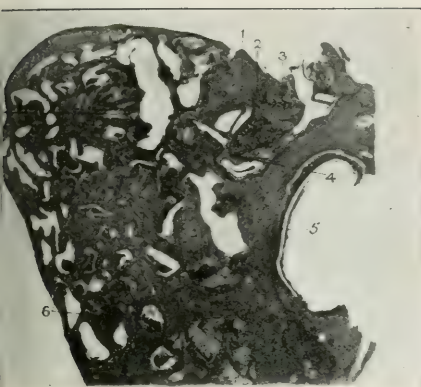


Fig. 29.

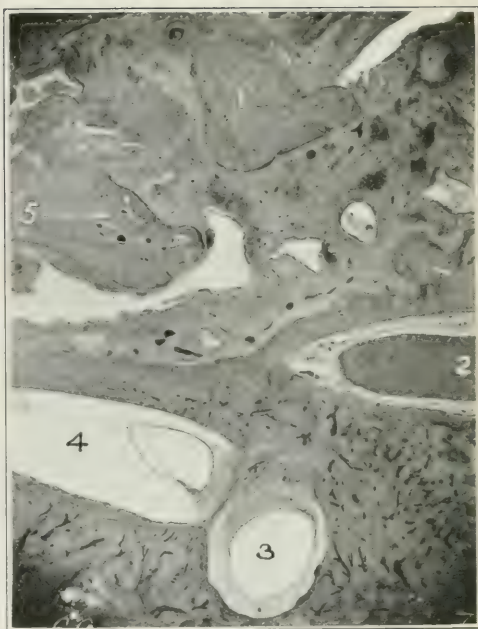


Fig. 30.

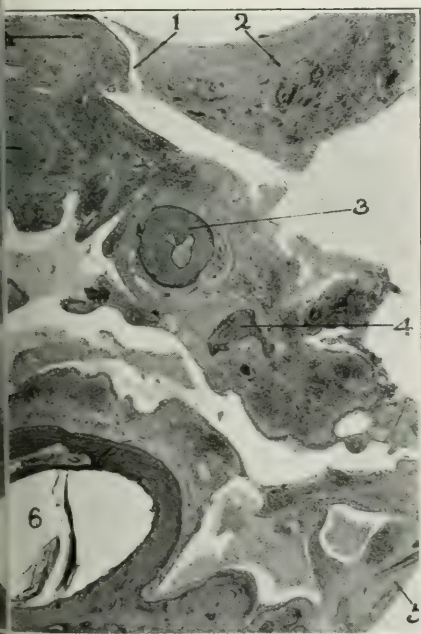


Fig. 31.



Fig. 32.

Fig. 33.—Case 1. Horizontal section through region of round window. 1, Engorged and infiltrated mucosa of promontory; 2, cochlear nerve in internal meatus, with meningitis; 3, hemorrhage in scala tympani; 4, hemorrhage in perilymph space of ampulla of posterior canal; 5, fracture between sinus tympani and crista of posterior canal; 6, exudate in niche of round window.

Fig. 34.—Case 2. Bullet wound of frontoparietal region, followed by intracranial hemorrhage. No direct injury to ear. Vertical section through cochlea, showing 1, hemorrhage in fundus of internal meatus around branches of cochlear nerve to the basal coil.

Fig. 35.—Case 2. Vertical section through vestibule and intravestibular part of cochlea. 1, Hemorrhage around nerve above vestibule; 2, facial nerve; 3, hemorrhage on inner wall of tympanum; 4, niche of oval window; 5, cochlear canal; 6, exudate in cochlear opening of perilymphatic aqueduct; 7, hemorrhage around nerve to ampulla of posterior canal.

Fig. 36.—Case 3. High explosive shell injury of ear. Horizontal section showing 1 and 2, everted edges of rupture in drumhead; 3, handle of malleus; 4, facial nerve with stapedius to left—the sinus tympani lies below; 5, posterior canal; 6, utricle; 7, internal meatus; 8, carotid canal; 9, opening of eustachian tube.

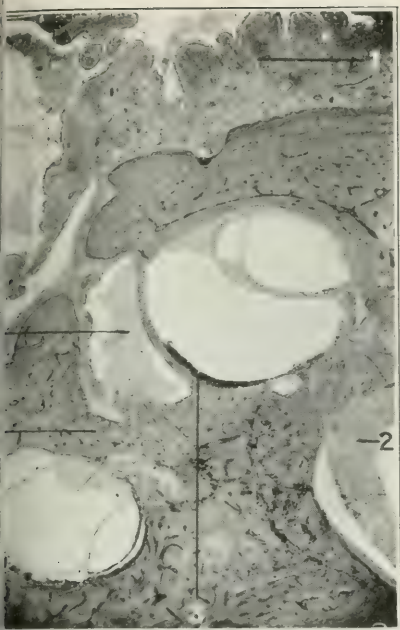


Fig. 33.



Fig. 34.

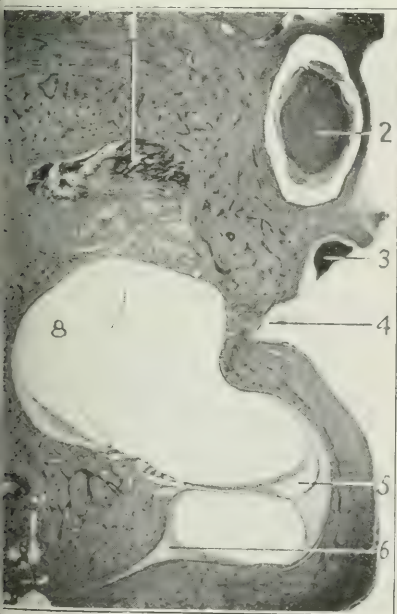


Fig. 35.



Fig. 36.

Fig. 37.—Case 4. Injury of ear due to explosion of rifle grenade. Horizontal section No. 125. 1, Facial nerve; 2, utricle; 3, detached otolith membrane of utricle; 4, ampullary end of lateral canal.

Fig. 38.—Case 4. Horizontal section through oval window, showing, 1, footplate of stapes; 2, neuroepithelium of saccule; 3, detached otolith membrane of saccule.

Fig. 39.—Case 4. Horizontal section through region of round window. 1, Rupture of drumhead; 2, scala tympani of basal coil of cochlea; 3, internal meatus with hemorrhage.

Fig. 40.—Case 4. Shows displacement of crista of lateral canal

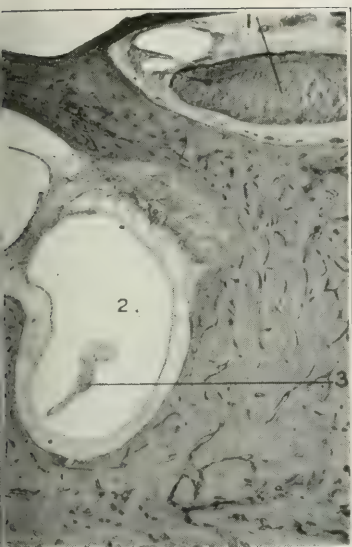


Fig. 37.



Fig. 38.

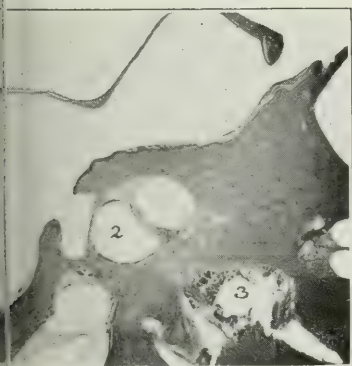


Fig. 39.

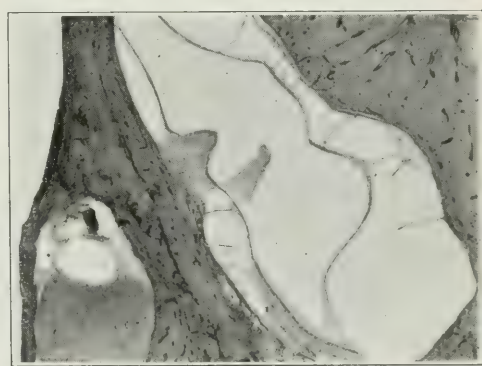


Fig. 40.

Fig. 41.—Case 6. High explosive shell injury of ear. Horizontal section through region of oval window. 1, Posterior part of rupture of drumhead; 2, handle of malleus with remains of drumhead attached; to the right the anterior part of the rupture is seen; 3, long process of incus; 4, hemorrhage in hollow of stapes; 5, facial canal; 6, chorda tympani nerve.

Fig. 42.—George Dick, aged 4. Chronic middle ear suppuration; erosion of lateral canal; cerebellar abscess. 1, Cholesteatoma covering prominence of lateral canal; 2, erosion of bone; 3, endolymph space; 4, perilymph space.

Fig. 43.—Donald Henry, aged 23. Chronic middle ear suppuration, right ear; temporosphenoidal abscess; meningitis. 1, Fistula in lateral canal; 2, perilymph space of lateral canal; 3, facial nerve; 4, erosion of bone.

Fig. 44.—Annie Bruce, aged 15. Chronic middle ear suppuration, right ear; cholesteatoma; latent labyrinthitis; perisinus abscess; sinus thrombosis. 1, Endosteum lining lateral canal; 2, endolymph space containing exudate; 3, granulation tissue in perilymph space; 4, facial nerve.

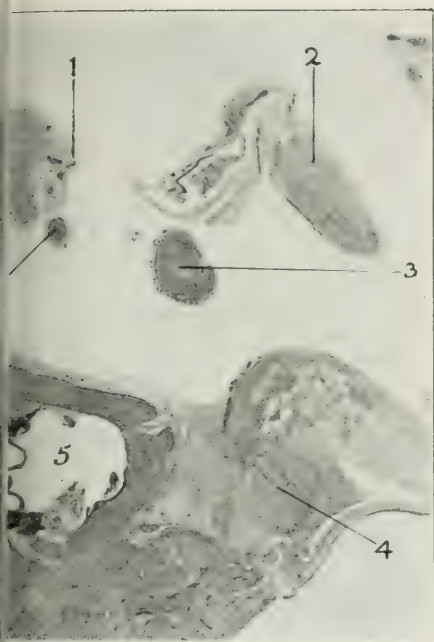


Fig. 41.

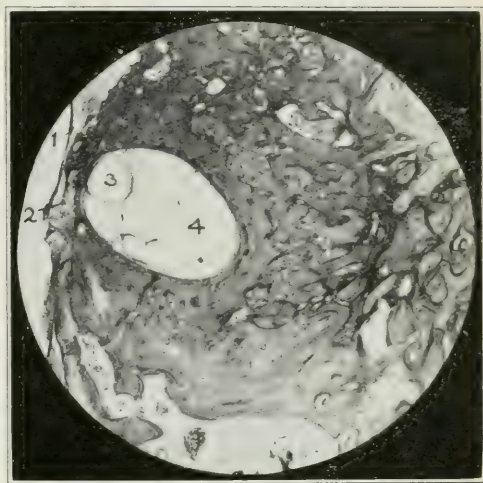


Fig. 42.

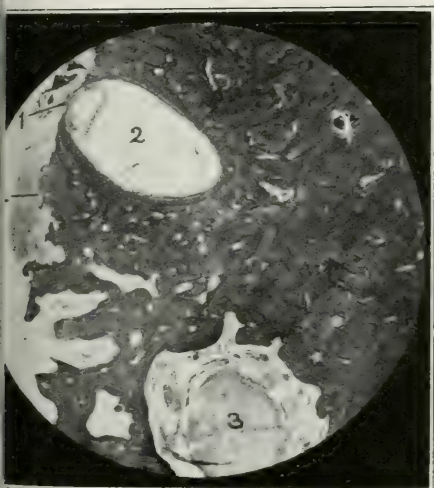


Fig. 43.

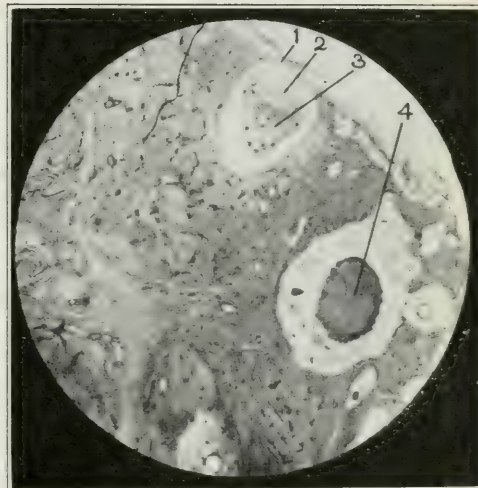


Fig. 44.

Fig. 45.—John Fisher, aged 11. Chronic middle ear suppuration, right ear; cholesteatoma; meningitis. 1, Prominence of lateral semicircular canal; 2 coagulated lymph in perilymph space of lateral canal (serous meningitis?); 3, smooth end of lateral canal; 4, facial nerve.

Fig. 46.—Same specimen as Fig. 45 under higher power. 1, Endolymph space, normal; 2, perilymph space containing coagulated fibrin.

Fig. 47.—John Johnson, aged 12. Acute middle ear suppuration (bilateral); vomiting; restlessness, with meningitic cry followed by coma. Right ear, showing hemorrhages into labyrinth but no labyrinthitis. 1, Tensor tympani; 2, purulent exudate in tympanum; 3, malleus; 4, incus; 5, exudate; 6, facial nerve—below the facial nerve there is hemorrhage into the perilymph space of the vestibule; 7, superior canal; 8, internal meatus with meningitis; 9, basal coil of cochlea with hemorrhage in scala vestibuli; 10, carotid canal.

Fig. 48.—John Johnson right ear. Basal coil of cochlea. 1, Hemorrhage in cochlear canal; 2, scala vestibuli; 3, spiral ganglion; 4, hemorrhage along nerve in bony spiral lamina; 5, scala tympani.

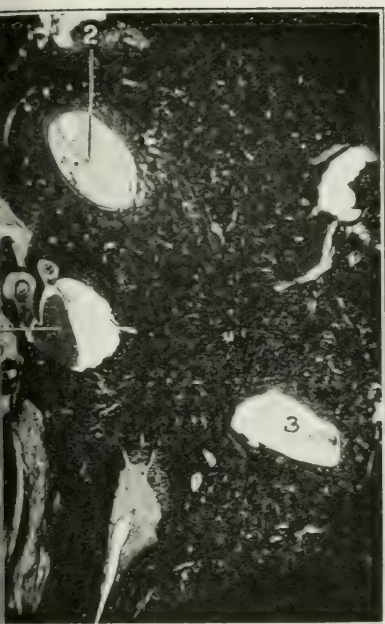


Fig. 45.



Fig. 46.



Fig. 47.

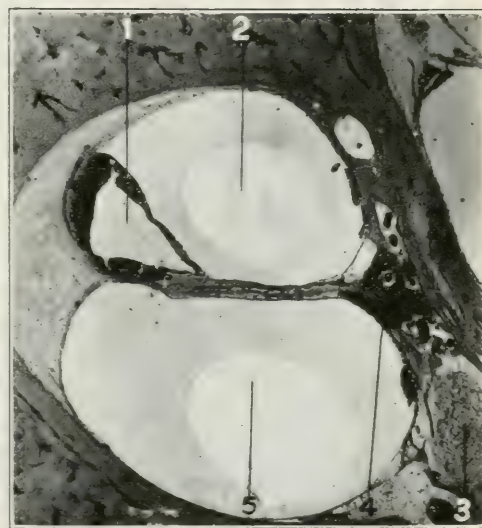


Fig. 48.

Fig. 49.—John Johnson, right ear. Horizontal section through oval window. 1. Hemorrhage external to sacculus; 2, pus in hollow of stapes; 3, footplate of stapes; 4 and 6, hemorrhage in vestibule; 5, utricle; 7, ductus endolymphaticus; 8, saccule.

Fig. 50.—John Johnson, right ear. Horizontal section through lower part of tympanum and labyrinth. 1. Pus in tympanic cavity; 2, external meatus; 3, joint between incus and stapes; 4, facial nerve; 5, posterior canal; 6, aqueduct of vestibule; 7, junction of perilymph space of vestibule with scala vestibuli; 8, hemorrhage in basal coil of cochlea.

Fig. 51.—John Johnson, right ear. 1, Scala tympani; 2, hemorrhage internal to round window membrane; 3, niche of round window lined by swollen mucosa; 4, membrane of round window; 5, pus in cochlear opening of perilymphatic aqueduct; 6, vein accompanying perilymphatic aqueduct.

Fig. 52.—John Johnson, left ear. On this side purulent labyrinthitis was present in addition to middle ear suppuration. Horizontal section through region of oval window. 1, External meatus; 2, incus; 3, malleus; 4, facial nerve; 5, swollen mucosa of tympanum; 6, basal coil of cochlea containing pus; 7, internal meatus; 8, pus in vestibule; 9, utricle; 10, opening of lateral canal into vestibule; 11, lateral canal; 12, posterior canal.

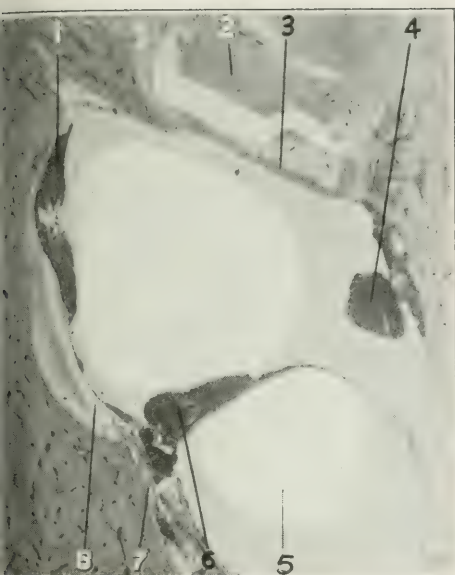


Fig. 49.

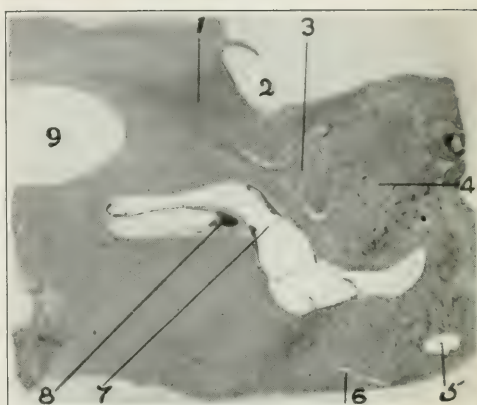


Fig. 50.

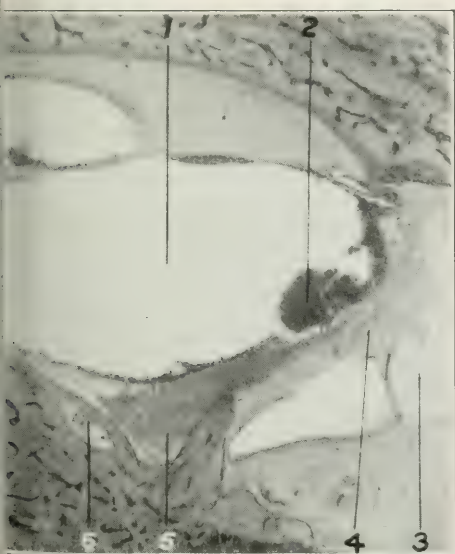


Fig. 51.

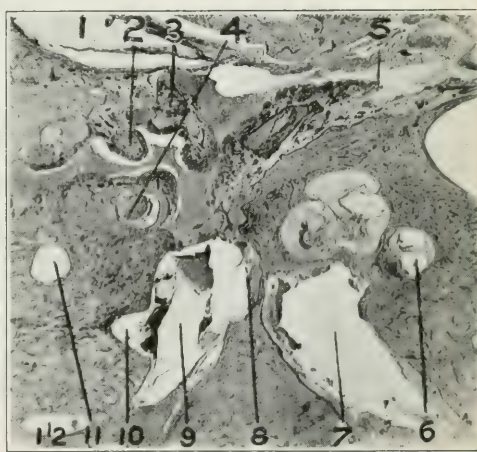


Fig. 52.

Fig. 53.—John Johnson, left ear. Horizontal section through oval window. 1, Swollen mucosa over facial canal; 2, pus in tympanic cavity; 3, footplate of stapes—notice the pus entering the vestibule at both edges of the footplate; 4, posterior crus of stapes; 5, internal meatus with meningitis; 6, ruptured saccule; 7 and 8, pus in vestibule; 9, utricle.

Fig. 54.—John Johnson, left ear. Section through drumhead, showing formation of perforation. 1, Chorda tympani; 2, external meatus; 3 pus in tympanum bursting through fibrous layer of drumhead; 4, desquamated epithelium of drumhead; 5, fibrous layer of drumhead; 6, promontory; 7, mucous membrane layer of drumhead; 8, pus in tympanum.

Fig. 55.—John Johnson, left ear. Horizontal section through region of round window. 1, Facial nerve; 2, stapedius; 3, external meatus; 4, swollen drumhead; 5, carotid canal; 6, basal coil of cochlea full of pus; 7, posterior part of membrane of round window; 8 and 9, two ends of posterior vertical canal.

Fig. 56.—John Johnson, left ear. Section through region of round window. 1, Pus in tympanum; 2, swollen mucosa of promontory; 3, pus in scala tympani; 4, vein accompanying aqueduct of cochlea; 5, cochlear opening of perilymphatic aqueduct; 6, pus in niche of round window—note that between 3 and 6 the membrane of the round window is disintegrated; 7, ampullary end of posterior canal.



Fig. 53.

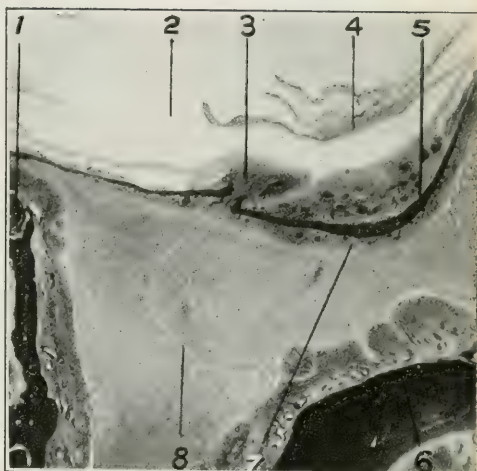


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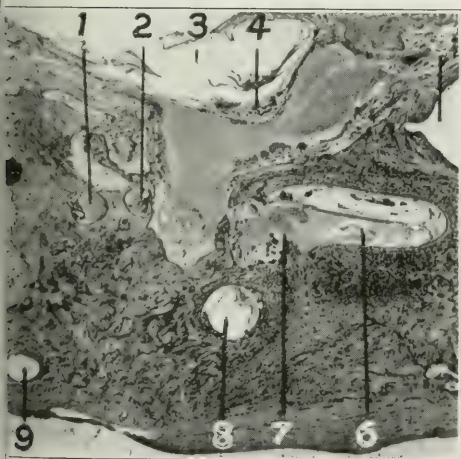


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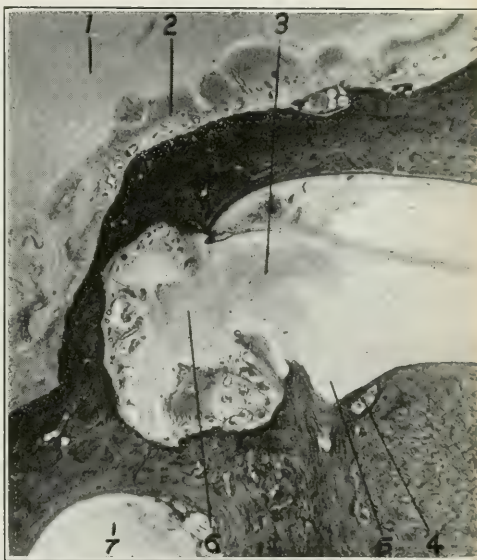


Fig. 56.

Fig. 57.—Mrs. Carson, aged 20. Chronic middle ear suppuration, right ear; vomiting, shivering and headache; giddiness; facial paralysis and third degree nystagmus to left. Vertical transverse section through oval window. 1, Facial canal; 2, thickened mucosa in niche of oval window; 3, pus in perilymph space of vestibule and scala vestibuli; 4, exudate in scala tympani; 5, footplate of stapes displaced into vestibule.

Fig. 58.—Mrs. Carson. Vertical transverse section through oval window. 1, Ampullary end of superior canal; 2, facial nerve; 3, oval window, empty; 4, thickened mucosa of round window; 5, ampullary end of posterior canal containing pus; 6, opening of crus commune.

Fig. 59.—Mrs. Carson. Section through basal coil of cochlea. 1, Fibrinous exudate in scala vestibuli; 2, dilated cochlear canal; 3, spiral ligament; 4, fibrinous exudate in scala tympani; 5, Corti's organ; 6, nerve in bony spiral lamina; 7, Reissner's membrane pulled inwards towards modiolus by contraction of exudate in scala vestibuli.

Fig. 60.—Thomas Gilmour, aged 17. Chronic middle ear suppuration, right; giddiness; death from cerebellar abscess. 1, Superior canal containing granulation tissue; 2, ampullary end of superior canal; 3, fistula in lateral canal; 4, facial nerve surrounded by granulation tissue; 5, polypus projecting into external meatus; 6, perforation of drumhead; 7, jugular bulb showing new connective tissue and bone formation; 8, ampullary end of posterior canal with pus; 9, lateral canal.

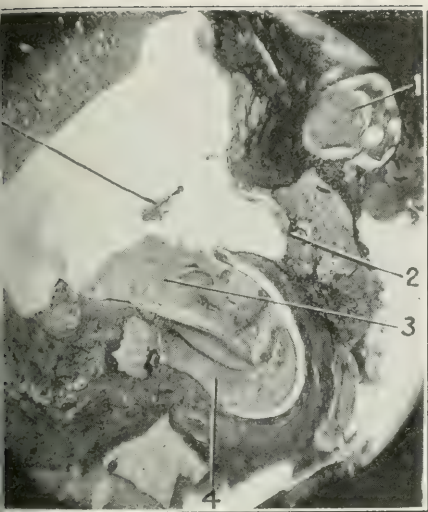


Fig. 57.



Fig. 58.

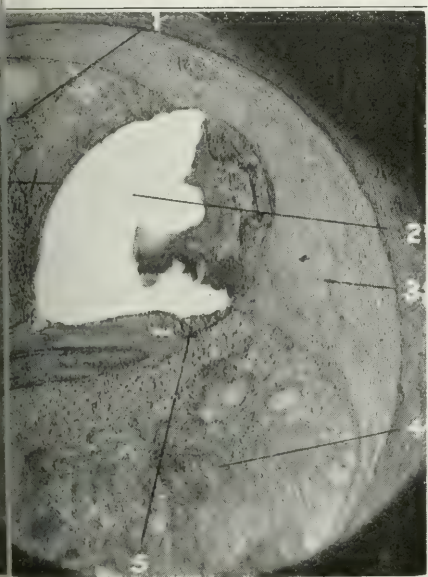


Fig. 59.



Fig. 60.

Fig. 61.—Thomas Gilmour. 1, Cholesteatoma in antrum; 2, polypus, which is originating in posterior cranial fossa; 3, stapedius; 4, lower part of posterior canal; 5, region of extradural abscess.

Fig. 62.—Christina Goodall, aged 6. Chronic middle ear suppuration, left ear; latent labyrinth suppuration; panlabyrinthitis; septicemia. Horizontal section through attic. 1, Outer attic; 2, polypus projecting from inner wall of attic; 3, cholesteatoma; 4, smooth end of superior canal; 5, ampullary end of superior canal, surrounded by area of granulation tissue; 6, basal coil of cochlea; 7, facial nerve.

Fig. 63.—Christina Goodall. Panlabyrinthitis. Horizontal section through upper part of vestibule and cochlea. 1, Osteomyelitis in promontory; 2, cholesteatoma in tympanum; 3, facial nerve; 4, lateral canal surrounded by granulation tissue; 5, cholesteatoma in mastoid; 6 and 7, engorged marrow spaces; 8, smooth end of superior canal containing pus; 9, pus in vestibule; 10, area of osteomyelitis; 11, vestibular nerve embedded in granulation tissue; 12, cochlear nerve; 13, basal coil of cochlea containing granulation tissue—note the dilatation of the cochlear canal.

Fig. 64.—Christina Goodall. Panlabyrinthitis. Section through lower part of vestibule and cochlea. 1, Cholesteatoma; 2, malleus; 3, incus; 4, lateral canal surrounded by granulation tissue (osteomyelitis); 5, crus commune in some condition; 6, footplate of stapes; 7, basal coil of cochlea filled by granulation tissue; 8, upper part of basal coil.

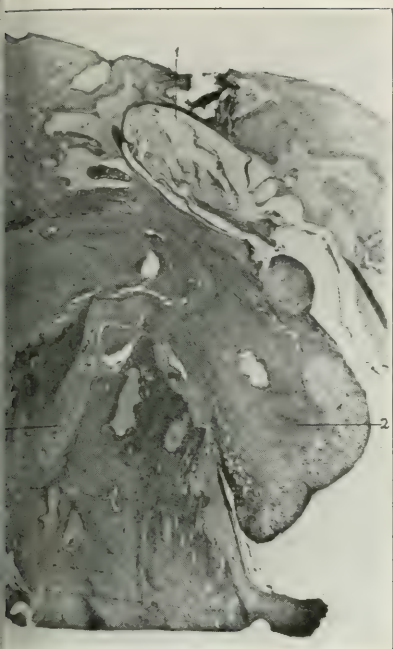


Fig. 61.

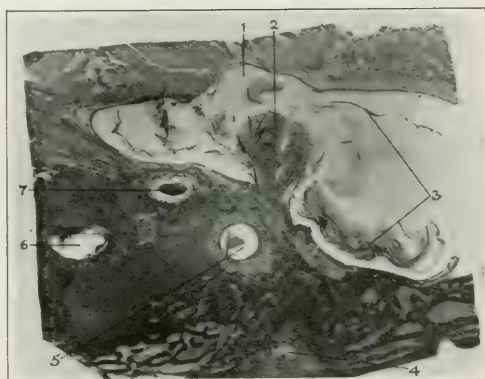


Fig. 62.

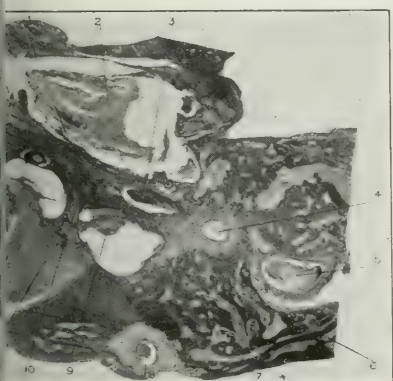


Fig. 63.

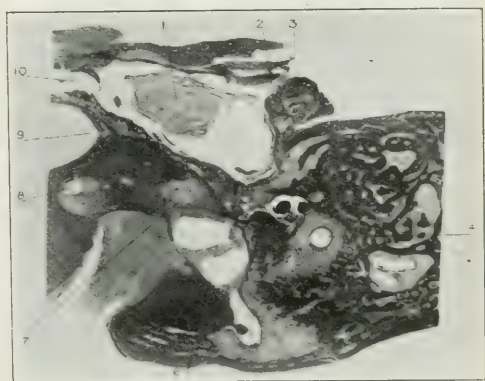


Fig. 64.

Fig. 65.—Christina Goodall. Panlabyrinthitis. Horizontal section through lower part of labyrinth. 1, New connective tissue in scala tympani internal to round window; 2, stapedius; 3 and 4, malleus and incus; 5, facial nerve; 6, opening of smooth end of lateral canal into vestibule—note the osteomyelitis around this and also the crus commune below it; 7, endolymphatic aqueduct; 8 and 9, areas of osteomyelitis; 10, new bone formation in scala tympani of basal coil; 11, carotid canal.

Fig. 66.—Walter Penman, aged 42. Chronic middle ear suppuration, left; giddiness; headache; insanity developed; death from endocarditis. Vertical transverse section through cochlea. 1, Internal meatus showing great thickening of dura and new fibrous tissue formation around nerves; 2, modiolus; 3, middle coil of cochlea with exudate; 4, tensor tympani; 5, tympanus; 6, carotid canal; 7, basal coil of cochlea filled up by new bone; 8, jugular bulb; 9, perilymphatic aqueduct.

Fig. 67.—Walter Penman. Vertical section through vestibule. 1, Extradural abscess between middle and posterior cranial fossae; 2, vestibule filled by connective tissue; 3, oval window; 4, facial nerve; 5, polypoid mucosa of tympanum; 6, malleus; 7, external meatus; 8, promontory; 9, niche of round window filled by connective tissue; 10, ampullary end of posterior canal; 11, jugular bulb.

Fig. 68.—William Newberry, aged 26. Chronic middle ear suppuration, left ear; cholesteatoma; sinus thrombosis; latent labyrinth suppuration; cerebellar abscess. Vertical transverse section through anterior part of cochlea. 1, Facial nerve; 2, internal meatus; 3, connective tissue in vestibule and basal part of cochlea; 4, region of round window; 5, promontory; 6, new bone formation.

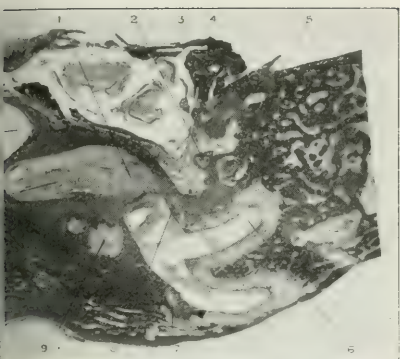


Fig. 65.

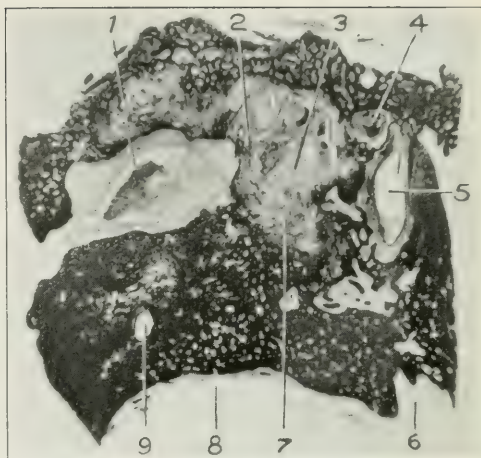


Fig. 66.

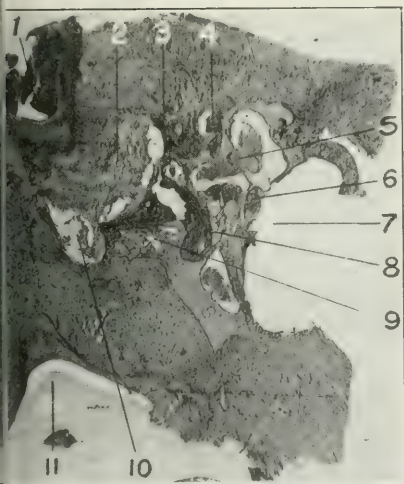


Fig. 67.

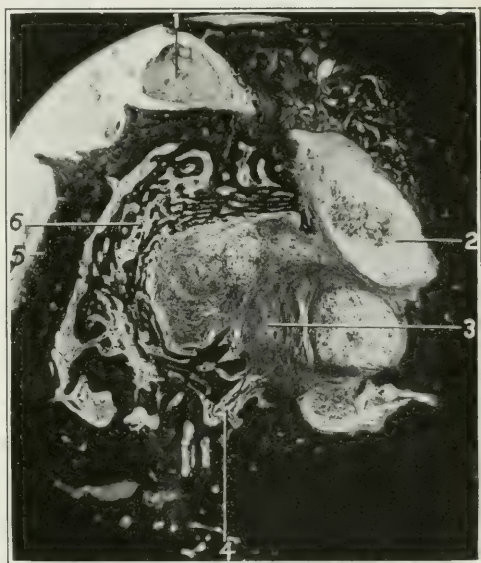


Fig. 68.

Fig. 69.—William Newberry. Vertical transverse section through oval window. 1, Facial nerve; 2, footplate of stapes displaced into vestibule, probably by contraction of fibrous tissue; 3, promontory; 4, round window membrane; 5, connective tissue in vestibule.

Fig. 70.—William Newberry. 1, New bone formation in vestibule; 2, new connective tissue formation; 3, abscess cavity; 4, niche of round window; 5, eroded promontory; 6, oval window; 7, facial nerve.

Fig. 71.—William Armstrong, aged 5. Chronic middle ear sup-puration, right ear; complete deafness; latent labyrinthitis passing on to spontaneous cure; death from meningitis after operation. Horizontal section through inner ear. 1, Remains of abscess in cochlea; 2, vestibule filled up by new bone; 3, posterior canal filled up by new bone; 4, aqueduct of vestibule; 5, internal meatus showing nerves surrounded by meningitis; 6, new bone formation in cochlea; 7, normal cartilage bone capsule of cochlea; 8, normal lamellar bone.

Fig. 72.—Margaret Wood, aged 52. Chronic middle ear sup-puration, right ear; spontaneous cure of labyrinthitis; thrombosis of sigmoid sinus and jugular vein; death from pyemia. Horizontal section through cochlea. The figure shows—1, that the whole of the cochlea has become a solid mass of bone; 2, internal meatus containing markedly atrophied nerves; 3, dilated saccule.

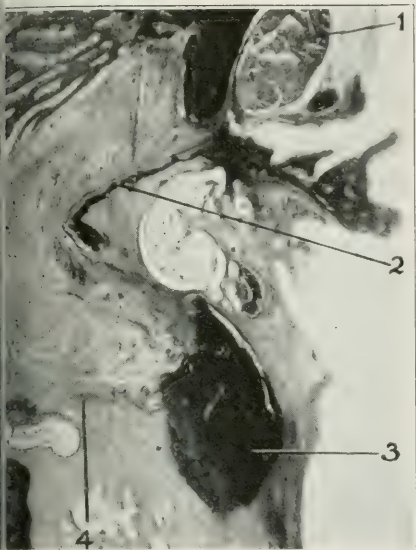


Fig. 69.

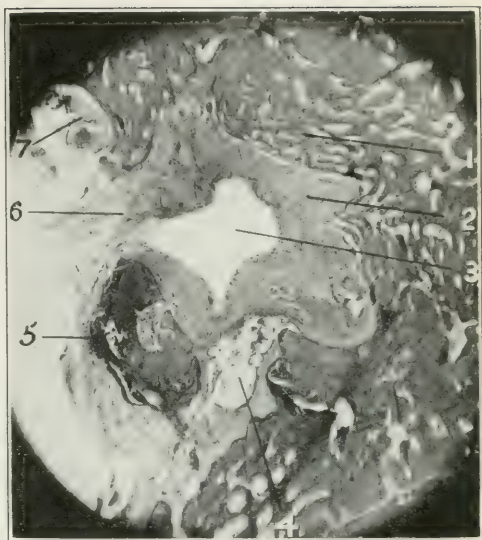


Fig. 70.

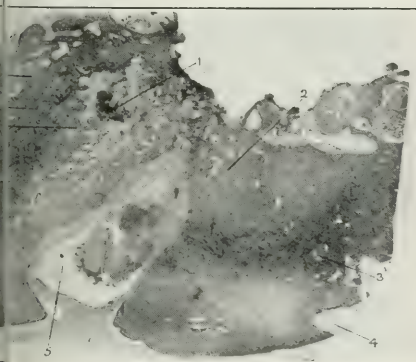


Fig. 71.

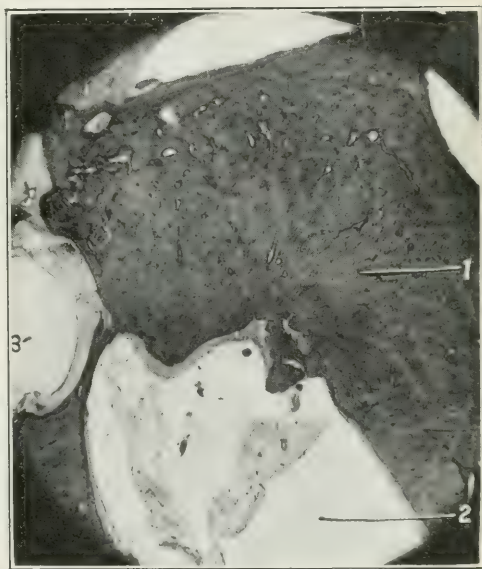


Fig. 72.

Fig. 73.—Margaret Wood. Horizontal section through oval window. 1, Facial nerve; 2, cystic space containing exudate; 3, footplate of stapes; 4, dilated saccule; 5, dilated utricle.

Fig. 74.—Case 1. W. Young, aged $1\frac{1}{4}$ years. Vertical transverse section of right ear, showing tubercular infiltration and erosion of walls of tympanic cavity. 1, Basal coil of cochlea; 2, tubal portion of tympanic cavity; 3, tensor tympani; 4, tubercular tissue in roof of cavity; 5, cochlear nerve in internal auditory meatus.

Fig. 75.—Case 1. Vertical transverse section, showing invasion of vestibule through oval window. 1, Membrane of round window; 2, tubercular erosion of promontory; 3, head of stapes; 4, tubercular tissue in niche of oval window; 5, facial nerve; 6, vestibular nerve to utricle, external and superior canals; 7, footplate of stapes eroded and displaced towards vestibule; 8, vestibular nerve to ampulla of posterior canal; 9, tubercular tissue filling up niche of round window.

Fig. 76.—Case 1. Vertical transverse section through canals. 1, Two ends of lateral canal; 2, fossa subarcuata infiltrated with tubercle; 3, two ends of posterior canal; 4, stapedius; 5, facial nerve; 6, erosion of bone over lateral canal prominence.

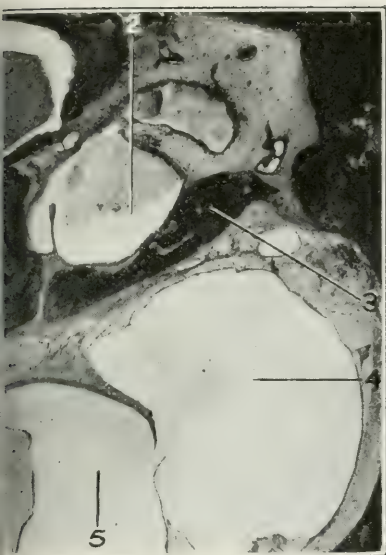


Fig. 73.

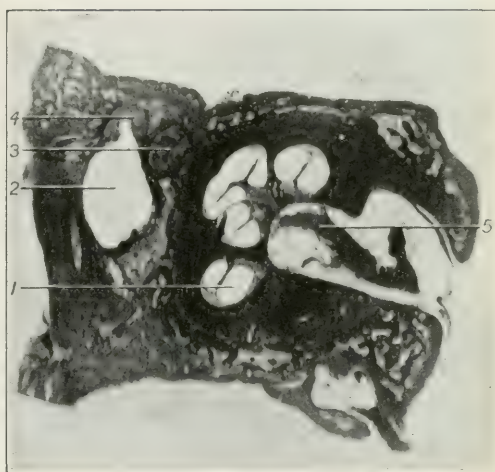


Fig. 74.

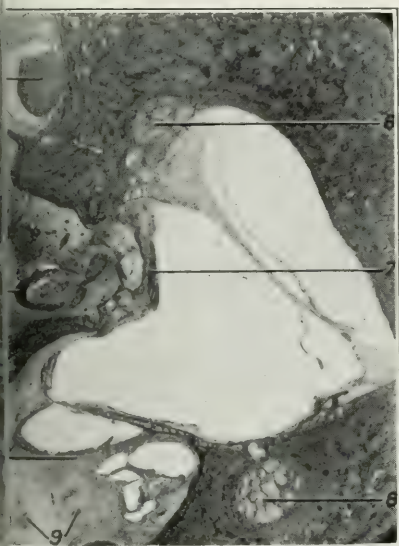


Fig. 75.

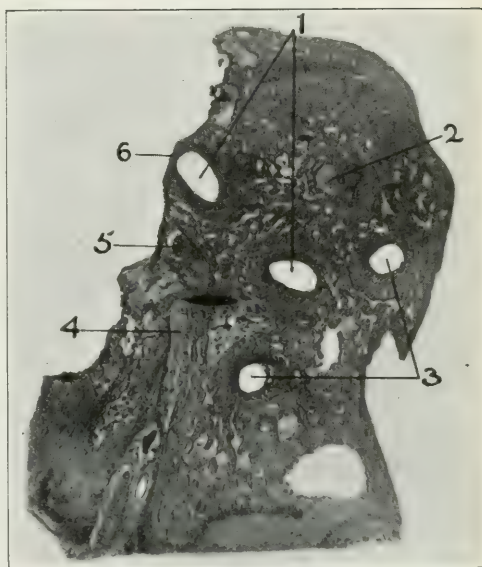


Fig. 76.

Fig. 77.—Case 2. J. Nisbet. Advanced tubercular disease of the ear. Vertical transverse section through apex of petrous bone. 1, Region of eustachian tube. The cartilaginous tube has disappeared. Cascating tubercle is seen to the left of and below the internal carotid artery. 2, Internal carotid; 3, apex of petrous bone; in this region the bone consists largely of marrow spaces.

Fig. 78.—Case 2. Vertical transverse section through anterior part of petrous pyramid. 1, Internal carotid artery; 2, tubercular granulation tissue infiltrating the wall of the carotid artery; 3, region of eustachian tube; the tube can no longer be recognized; 4, anterior part of petrous pyramid.

Fig. 79.—Case 2. Vertical transverse section through cochlea. 1, Fistula into basal coil of cochlea; 2, middle coil of cochlea—all scalae are filled with tubercular granulation tissue; the osseous spiral lamina is still to be seen; 3, fistula into apex of cochlea; 4, cartilage bone capsule of cochlea; 5, lamellar bone surrounding cochlear capsule; 6, tubercular pachymeningitis in floor of middle cranial fossa; 7, facial nerve; 8, auditory nerve; 9, cellular infiltration in fundus of internal meatus.

Fig. 80.—Case 2. Vertical transverse section through semicircular canals. 1, Erosion of bone by tubercular granulation tissue; 2, ampullary end of superior canal; 3, tubercular granulation tissue in fossa subarcuata; 4, smooth end of superior canal (perilymph space); 5, endolymphatic space of smooth canal; 6, saccus endolymphaticus (normal); 7, smooth end of posterior semicircular canal with granulation tissue.



Fig. 77.

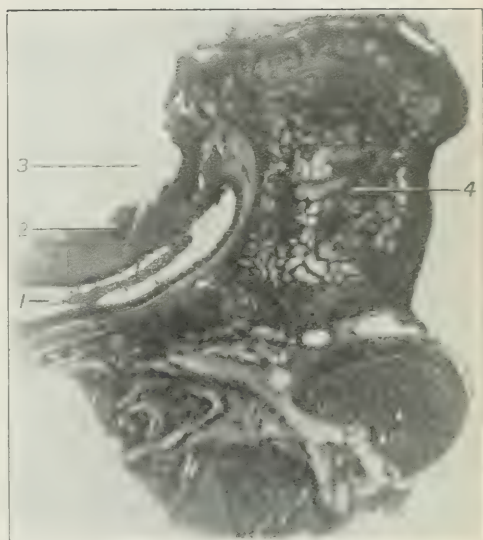


Fig. 78.

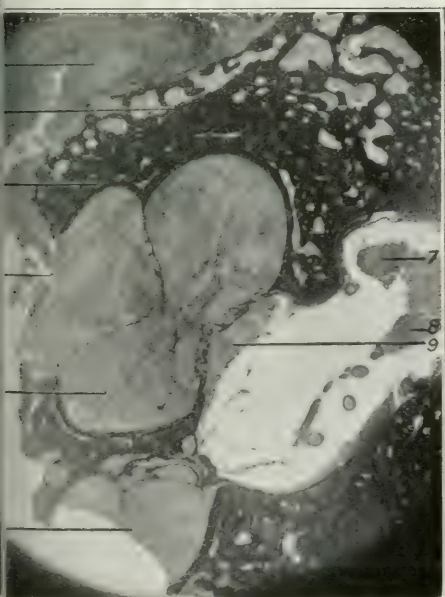


Fig. 79.



Fig. 80.

Fig. 81.—Case 3. John Cunningham. Vertical section through cochlea. 1, Facial nerve; 2, dilated scala media of middle coil; 3, fistula between scala vestibuli of basal coil and tympanum; 4, basal coil of cochlea filled up by new connective tissue and bone; 5, internal meatus with meningitic infiltration; 6, new formed bone filling up scala vestibuli.

Fig. 82.—Case 3. Section through middle coil of cochlea. 1, Bony spiral lamina; 2, Reissner's membrane pulled inwards by contraction of connective tissue; 3, dilated cochlear canal; 4, small celled infiltration; 5, basilar membrane; 6, remains of Corti's organ; 7, scala tympani filled by connective tissue.

Fig. 83.—Case 3. 1, New connective tissue and bone in wall of vestibule; 2, middle cranial fossa; 3, facial nerve; 4, dehiscence in facial canal; 5, large fistula into vestibule in region of oval window; the promontory has disappeared; 6, tubercular sequestrum in lower part of vestibule.

Fig. 84.—Case 3. 1, New formed bone partially filling up vestibule and region of ampullary end of posterior canal; 2, middle cranial fossa; 3, dehiscence in facial canal—caused by tubercular erosion (?); 4, tubercular granulation tissue in center of vestibule.

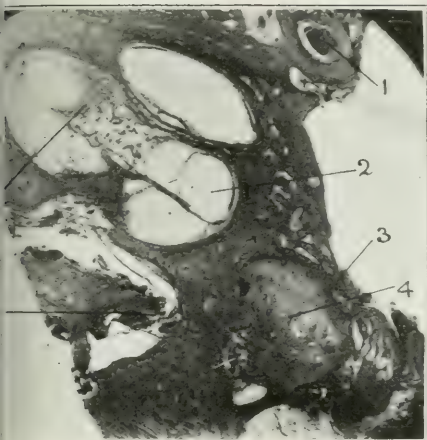


Fig. 81.

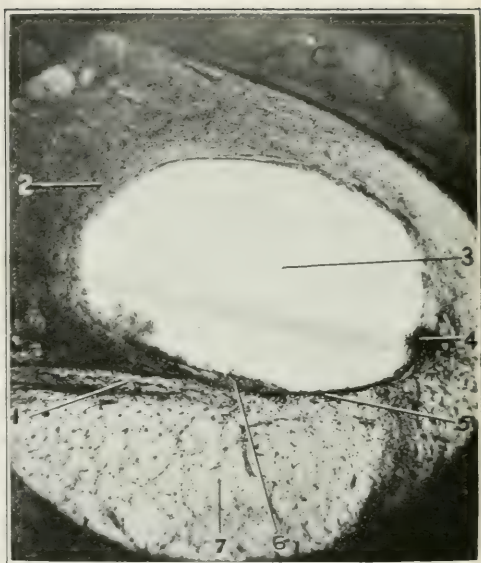


Fig. 82.

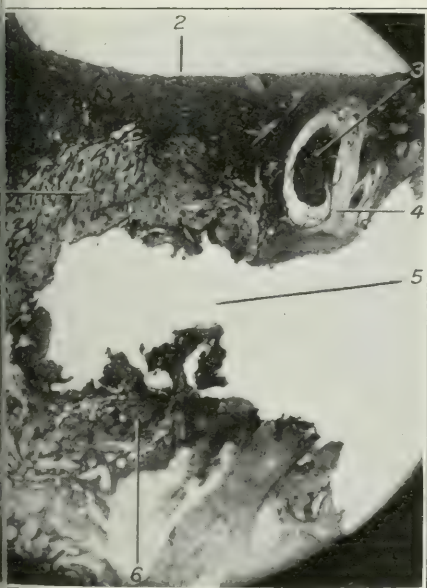


Fig. 83.

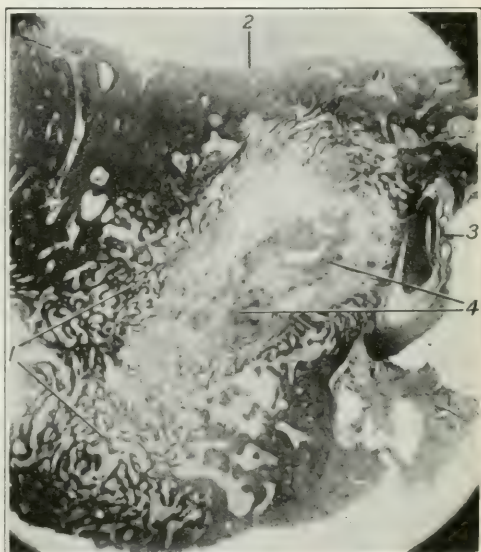


Fig. 84.

Fig. 85.—A case with Meniere's symptoms and facial paralysis. Vertical section of cochlea and internal auditory meatus. 1, Small cell infiltration in internal meatus; 2, above the seventh nerve; 3, beneath the epineurium.

Fig. 86.—Case 1. Tumor of right nervus acusticus. Horizontal section of right ear. Axial section through cochlea and internal meatus. 1, Scala media of upper part of middle coil, showing Corti's organ; 2, new bone formation in scala tympani; 3, saccule; 4, new connective or tumor tissue in scala tympani of basal coil; 5, erosion of cartilage capsule of cochlea by tumor tissue; 6, cochlear canal of lower part of basal coil, showing absence of Corti's organ and degeneration of spiral ligament.

Fig. 87.—Case 1. Horizontal section through basal coil of cochlea (right ear). 1, Reissner's membrane; 2, spiral canal with marked atrophy of ganglion cells; infiltration by delicate connective tissue; 3, delicate connective or tumor tissue in scala tympani; 4, large vein; 5, new formed bone; 6, myxomatous or dropsical degeneration of spiral ligament; 7, stria vascularis—note absence of Corti's organ.

Fig. 88.—Case 1. Horizontal section of right ear. 1, Tympanic membrane; 2, serous exudate in niche of round window; 3, connective or tumor tissue in scala tympani of basal coil; 4, cochlear nerve in fundus of internal meatus; 5, site of tumor which has been removed; in the right hand bottom corner a piece of the tumor tissue still remains; 6, aqueduct of cochlea and accompanying vein; 7, two ends of posterior canal (note that the perilymph space at the smooth end is almost filled by connective tissue); 8, serous exudate in air cells.



Fig. 85.

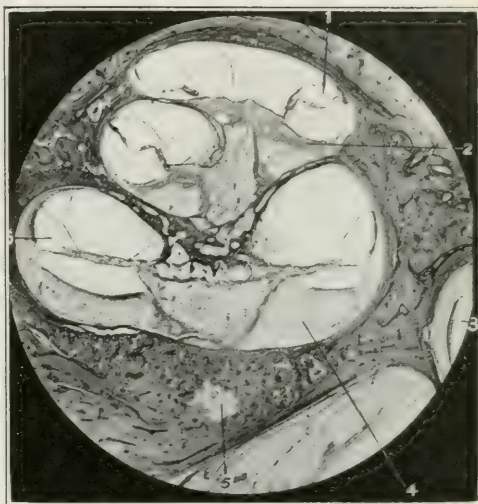


Fig. 86.

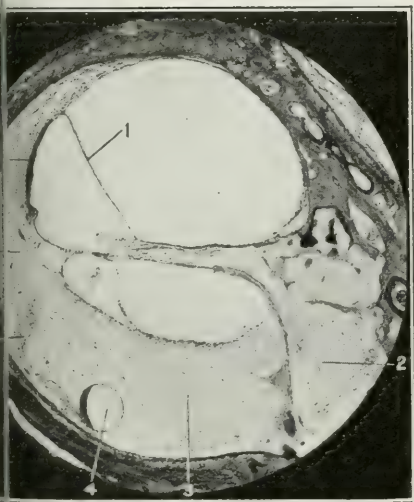


Fig. 87.

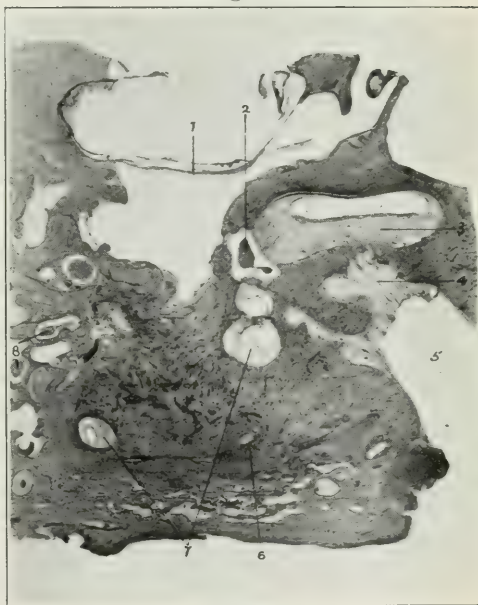


Fig. 88.

Fig. 89.—Case 1. Horizontal section of left ear. New connective tissue in scala tympani of apical coil; 2, greatly dilated cochlear canal of upper part of middle coil; note new connective tissue and bone in scala vestibuli; 3, spiral ganglion cells still present; 4, cochlear nerve; 5, hemorrhage between dura mater and bone; 6, scala tympani of lower part of basal coil with new connective tissue and bone formation; 7, scala vestibuli of middle coil filled with new bone formation.

Fig. 90.—Case 2. Tumor of eighth nerve. Horizontal section of right ear. 1, Upper part of basal coil of cochlea; 2, facial nerve—the tumor tissue is infiltrating as far as the geniculate ganglion; 3, tumor infiltrating vestibular nerve to utricle; 4, utricle; 5, facial nerve; 6, convexity of lateral canal; 7, smooth end of posterior canal; 8, erosion of bone by tumor; 9, crus commune; 10, tumor.

Fig. 91.—Case 2. Horizontal section of right ear. 1, Head of stapes with stapedius; 2, facial nerve; 3, posterior canal with hemorrhage in perilymph space; 4, tumor; 5, lower part of utricle; 6, dilated internal meatus with tumor.

Fig. 92.—Case 2. Horizontal section of right ear. 1, Thickened membrane of round window; 2, new bone formation in connective or tumor tissue in scala tympani; 3, spiral canal infiltrated and ganglion cells absent; 4, remains of cochlear nerve in fundus of internal meatus; 5, canal for nerve to crista of posterior canal infiltrated as above; 6, endolymph space of posterior canal.



Fig. 89.



Fig. 90.

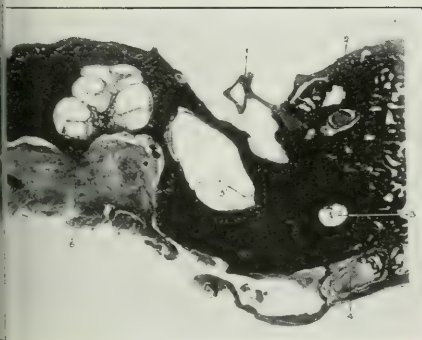


Fig. 91.

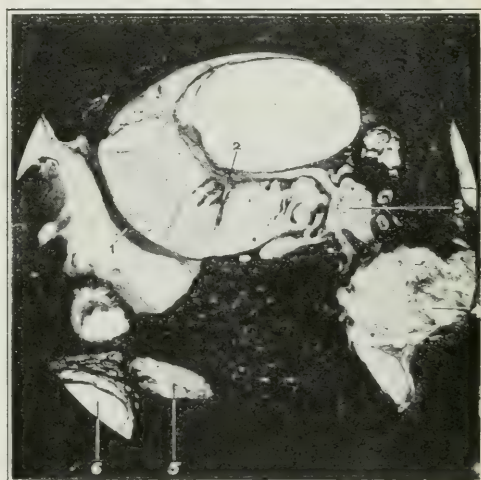


Fig. 92.

Fig. 93.—Case 2. Shows tumor of the pia arachnoid, which occupied one of the small bony erosions. The section shows a fibrous, cellular growth, apparently composed of neuroglial tissue. The neuroglial fibers appear to run in bundles in various directions. Cushing holds that these swellings are hypertrophied Pacchionian bodies, while Key believes that they are gliomatous metastases in the arachnoid. They certainly appear to be of a gliomatous nature.

Fig. 94.—Case 3. Horizontal section of right ear. 1, External meatus; 2, malleus; 3, tensor tympani; 4, gasserian ganglion; 5, tumor; 6, dilated saccule with granular contents; 7, utricle; 8, smooth end of posterior canal; 9, lateral canal; 10, footplate of stapes; 11, long process of incus.

Fig. 95.—Case 3. Axial section through right cochlea. 1, Dilated cochlear canal, basal coil; 2, capsule of tumor; 3, tumor tissue; 4, cochlear nerve compressed by tumor tissue; 5, central canal of modiolus.

Fig. 96.—Case 3. Axial section through right cochlea. 1, Fibrinous and granular exudate in scala vestibuli; 2, dilated cochlear canal; 3, stria vascularis; 4, fibrin threads in scala tympani; 5, spiral canal; some ganglion cells are still to be seen; 6, nerve entering bony spiral lamina.

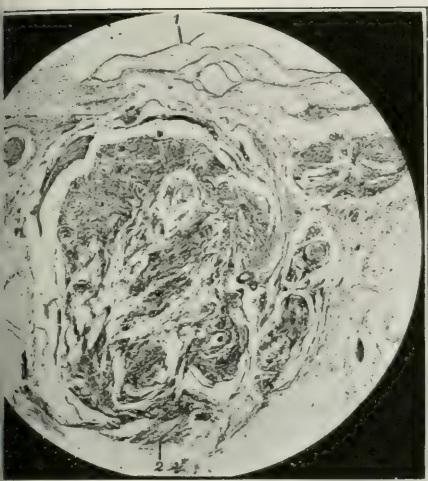


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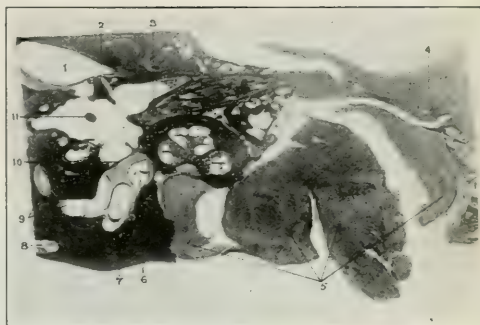


Fig. 94.

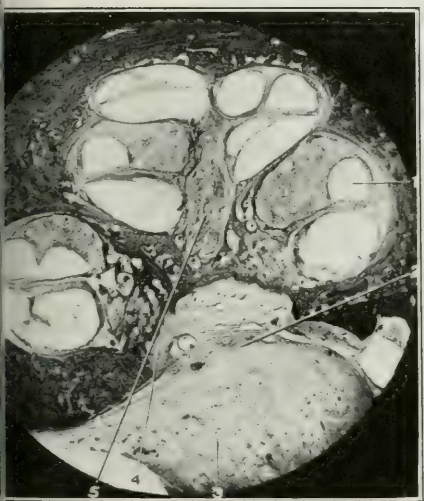


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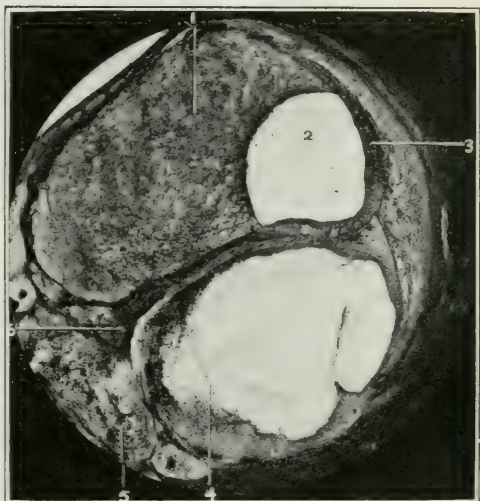


Fig. 96.



Fig. 97.

Fig. 97.—Case 3. Horizontal section of right ear. 1, External meatus; 2, hemorrhage in tubal portion of tympanic cavity; 3, internal carotid artery; 4, tumor; 5, cochlear opening of perilymphatic aqueduct; 6, region of saccus; 7, two parts of posterior canal.

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LXXI.

OBSERVATIONS OF ADENOIDS REMOVED WITH A DIRECT VISION ADENOTOME.

BY I. D. KELLEY, JR., M. D.,

ST. LOUIS.

Since Wilhelm Meyer¹ directed the profession's attention to adenoids, various instruments for their removal have been devised. The most popular in use are perhaps the Gottstein, Beckmann and Hartmann curets, the adenotomes or nasopharyngeal tonsillotomes of La Force and Schutz-Passow, and the Jurasz adenoid biting forceps. Each of these instruments employs a blind technic in adenoid removal.

From time to time, efforts have been made to remove adenoids under direct vision, sometimes by means of a palate retractor held in the left hand while the operation is done with the right hand. Recently, Joseph C. Beck² has employed rubber catheters passed through the nostrils and out of the mouth, which are tied, serving in this way as an elastic palate retractor. None of these efforts in my experience have been so satisfactory as the removal by the direct vision adenotome. Observation on the cadaver showed that a straight line was possible from the eye of the observer to the upper limit of the adenoid with this instrument in position. This is easily demonstrable on the living subject and has been the incentive in the development of this instrument and its use.

Figures 1 and 2 show the direct vision adenotome. The instrument consists of a hollow shaft or hood, which permits direct vision and serves as a soft palate retractor, the distal or blade end of which, when put into position, conforms to and rests directly against the posterior nasopharyngeal wall. From the proximal end of the shank extends a hand grip, placed at an angle that will prevent chin or chest interference.

when gripped and placed in position. On the upper surface of the shank is a shaft, to which is attached a flexible blade traversing the cutting surface at the distal end of the hood from below upward, thus permitting adenectomy under the vision of the surgeon.

The blind technic previously employed in adenoid removal is responsible for much of the diversity of opinion regarding adenoids found among operators in our clinics, which the routine use of the direct vision adenotome will help to make clear. When adenoids are removed with this instrument, the nasopharynx is encircled sufficiently by the cutting surface to show the adenoids entirely surrounded and presenting a definitely organized appearance, easily differentiated from the surrounding mucous membrane. The instrument can be so exactly placed about the adenoid, and such definite pressure exerted on the soft structure of the posterior wall, that the blade, when it engages the adenoid, is forced behind, severing it with its capsule. This exactness in technic, which is due to direct vision, has proved so unfailing that a large number of adenoid cases, checked by a careful and searching digital examination of the nasopharynx after operation, have shown the postnasal space entirely freed from lymphoid tissue. A removed adenoid has been likened, because of its supposedly loose construction, to the leaves of a poorly bound book spread apart and suspended, causing the leaves immediately to fall and scatter. As a matter of fact, my experience has been that adenoids removed with such precision as this instrument affords are quite well organized, owing, no doubt, to the presence on their cut surface of a definite fibrous capsule.

This ability to bring the adenoid into complete vision, coupled with the constant finding of a definitely organized mass of lymphoid tissue, indicates clinically that adenoids have a definite location in the nasopharynx. When we find by digital examination what seems to be a wild proliferation of adenoid tissue filling the postnasal space, it is by mass extension of the lymphoid units composing the adenoid and not the actual spread or proliferation of its basal attachment beyond easily recognized limits.

Barnes³ states, in his description of the location of adenoids, that the larger central adenoid mass occupies the median portion of the posterior wall with scattered lymphoid masses, extending into Rosenmüller's fossa, often filling it. I believe that if we embrace all lymphoid tissue found in this region under the term "adenoids," we should not only include the lymphoid tissue in Rosenmüller's fossa, which is an extension upward of that part of Waldeyer's ring known as the posterior lymph column, but must include as well the small isolated lymph patches on the posterior wall. This term cannot embrace all these structures, because in adenoids removed with direct vision we are dealing with a definitely located encapsulated mass of lymphoid tissue, which I feel is synonymous with the nasopharyngeal tonsil, and which is shown to have its attachment extending above to the posterior superior border of the nasal septum, below to the belly of the superior constrictor muscle, and laterally to the posterior lymph columns, found in hypertrophied adenoids as compared to the smaller atrophic organized adenoids occupying a lesser portion of this area.

Figure 3 represents two views of an adenoid which was completely surrounded and removed in direct vision. On the anterior or presenting surface are seen the several units composing the adenoid, represented by the crypts surrounded by eminences of lymphoid tissue. On the posterior or capsular surface is seen a well organized fibrous capsule with nodules and depressions similar to those found on the tonsil. Figures 4 and 5 are sections taken at random from many adenoids removed with their capsules, and show their histologic structure under low magnification. In the lowest magnification is seen the same histologic structure as the tonsil. At the top is a well developed capsule from the tunica propria of the mucous membrane with definite fibrous trabeculae descending into the lymphoid mass, composed of reticular connective tissue containing countless leukocytes in its meshes, and scattered throughout are the germinal centers or oval rings of lymphocytes. The higher magnification shows more clearly the tunica propria or capsule on which the adenoid is developed, its numerous mucous glands and blood vessels, together with the

trabeculae forming the coarser framework of the lymphoid tissue. The capsular structure is seen to be so well marked that it must serve to show that the previous methods of adenoid removal were responsible for much of the seeming lack of organization found in this tissue.

The direct vision adenotome permits a study of the adenoid immediately before and during removal. Many interesting forms of adenoid inflammation are seen, from simple blocking of the crypts with debris to suppurative follicular adenoiditis.

Figure 6 represents one of several adenoids removed where pus was seen to exude from a small opening on the adenoid surface; and while the adenoid was being severed from its attachment a long, wormlike mass of debris exuded from the opening of the pharyngeal bursa, partially filling the hood of the adenotome. In the left view is seen the presenting or anterior adenoid surface with a bristle inserted into the opening; while to the right is the posterior surface with the capsule partially removed, revealing multiple abscesses occupying the body of the adenoid. Figure 7 is a section through the adenoid shown in Figure 6, above the level of the abscesses, showing the pus track occupying the centers of the largest adenoid mass; while Figure 8 is a section through the multiple abscesses occupying the body of the adenoid. In the section at the top is also seen a portion of the adenoid capsule with its trabeculae descending toward the epithelial surface.

Thornwaldt⁴ describes a disease of the adenoid in which pus is seen to exude from the median bursa that has partially closed, as the result of inflammation, and the symptoms ascribed to this disease are persistent and recurrent attacks of pharyngitis. The clinical findings in the case are shown in Figures 6, 7 and 8, at operation under direct vision; and because of the large quantity of pus expelled from the adenoid they would suggest much more than purulent infection of a partially closed bursa described in Thornwaldt's disease. This, together with the extensive involvement shown on microscopic examination, could no doubt produce the same symptoms caused by pus absorption from the tonsils, teeth or focal pus infection elsewhere in the body.

Figure 9 is inserted to show that the median adenoid bursa is not a necessary factor in the production of these suppurative adenoids. The left view of Figure 9 is the presenting surface of an adenoid with an opening containing a bristle to the side of the median bursa, from which at operation an unusually large quantity of pus was expressed. To the right is the capsular surface with the capsule seen to be removed intact, with a protruding eminence in which pus was seen through the glistening semitransparent membrane. Figure 10 is a section through the same adenoid, showing the extensive involvement of the tissue surrounding the cavity. The cavity is surrounded by a wide necrotic area in which much of the lymphoid structure is destroyed.

When no adenoids are present, it is possible with the direct vision adenotome to study the smooth, glistening, velvet-like mucous membrane of the superior postnasal wall in contrast to the mucous membrane below covering the superior constrictor muscle which forms, when the superior constrictor is contracted, a raised border representing the upper edge of the muscle extending horizontally across the posterior nasopharyngeal wall, with pendicular rugæ running into the pharynx. It is this upper raised border of the muscle which is cut into and the rugæ stripped during the curette operation when inspection reveals tags hanging in the throat after adenoid removal.

The technic of adenoid removal with the direct vision adenotome is as follows:

Under local or general anesthesia, the mouth is widely opened with a gag; the instrument is inserted in the mouth; the posterior portion of the tongue is depressed with the under surface of the hood until above the upper surface is seen the free margin of the soft palate; the instrument is then gently pushed back to the posterior wall, and the distal or hood end is raised into the nasopharynx, the small ridge on the edge of the upper hood surface automatically engaging the soft palate border, forcing it forward on the surface of the hood out of harm's way. The distal end of the instrument is then further raised into the nasal vault until the adenoid is seen to be com-

pletely surrounded, when one looks through the hood, and in full view of the operator. The instrument is then pressed firmly against the posterior wall, and the razor sharp blade pushed closed by means of the thumb plate. The adenoid is thereby completely severed from its attachment, the operator seeing the blade cutting through the adenoid mass. When the instrument is removed, the adenoid will be found in a cup formed by the blade closing the inside of the hood.

Indications for the direct vision operation are found in all cases in which a complete surgical adenoid removal is desired. There are no contraindications to its use. Occasionally we find deep depressions on the posterior wall above the second cervical vertebra which the blade of this instrument, running as it does in a rigid arc, cannot reach; but this cannot be advanced as a contraindication, because even the blade of a curette used in the older operation is fixed between two rigid arms and, because of their interference, must traverse in its descent the same rigid arc. Therefore, to remove the remaining adenoid tissue in these depressions it is necessary to use a narrow curet, such as the one designed for such purposes by Sluder² (Figure 11), to reach these depressions, or biting forceps, which can best be done under direct vision through the hood of the direct vision adenotome. In several hundred observations we have found these depressions in only two cases. In cases in which the vault of the nasopharynx is unusually high, the use of the direct vision adenotome again has no contraindication if the technic of its introduction into the nasopharynx is properly carried out by feeling the definite resistance offered by the posterior superior border of the nasal septum and seeing the adenoid in complete vision.

When fragments of adenoid tissue are found not to have been removed from the vault, the resistance of the soft palate muscles is mistaken by the operator for the resistance felt when the upper distal border of the instrument comes in contact with the posterior superior border of the septum. Consequently, the vault has not been reached or the adenoid has not been completely surrounded and in full vision, as sometimes happens when the palate is not relaxed. Also, if the

operator releases his wrist tension when pushing the blade closed, the palate tension will force the cutting surface of the hood into a lower position on the posterior wall, thus permitting an upper unsevered portion of the adenoid to remain in the nasopharynx. With careful scrutiny of the adenoid in regard to complete vision during the cutting process, this error in technic should not occur.

Experience has shown in cases in which both tonsils and adenoids are to be removed that the direct vision adenotome should be used first, because the field of operation is unobscured. The "Sluder tonsillectomy" depends so little on the sense of sight, when properly understood, that a smear of blood in the pharynx is negligible. Therefore, removal of the adenoid should be the first operation done. However, in those unusual cases in which the tonsils are so large that the introduction of the adenotome into the nasopharynx is impossible, the tonsil should be first removed; the momentary gush of blood from the two operated fields is then allowed to stop, and the direct vision adenotome then introduced. Now, if the inside of the hood is sponged free of blood, full vision is permitted to complete the adenoid operation.

Frequently in clinics the use of the direct vision adenotome will reveal a wrong diagnosis. Cases are found in which the presence of adenoids is not noticed and the tonsils alone advised removed. Moreover, adenoids are often diagnosed when their presence does not exist. Operation under these conditions results in needless destruction of normal nasopharyngeal mucous membrane. Furthermore, with the routine diagnostic use of this instrument, it is surprising with what frequency adenoids are found in adults.

The use of the direct vision adenotome is advised as a diagnostic procedure in all tonsil and adenoid operations.

HUMBOLDT BUILDING.

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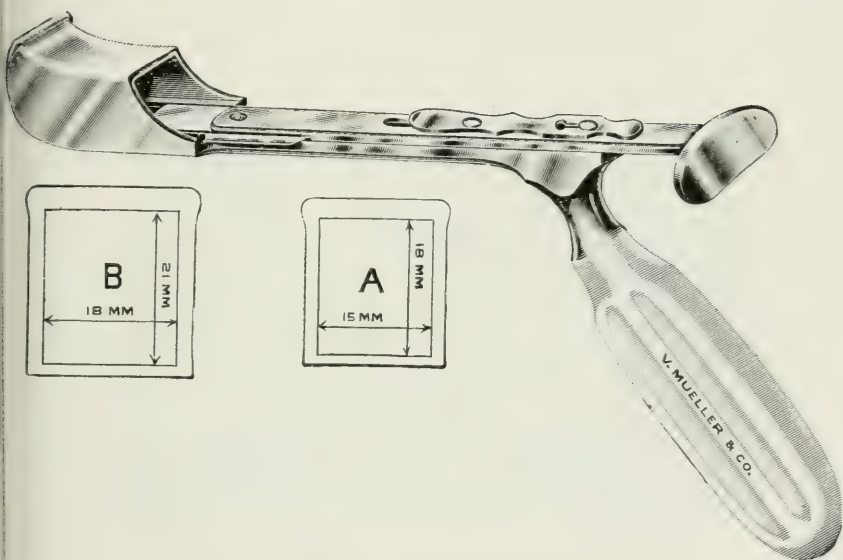


Fig. 1. Direct vision adenotome: A, dimensions of small instrument, to be used in children approximately under five years of age; B, dimensions of larger instrument, to be used in adults and children over 5 years of age.

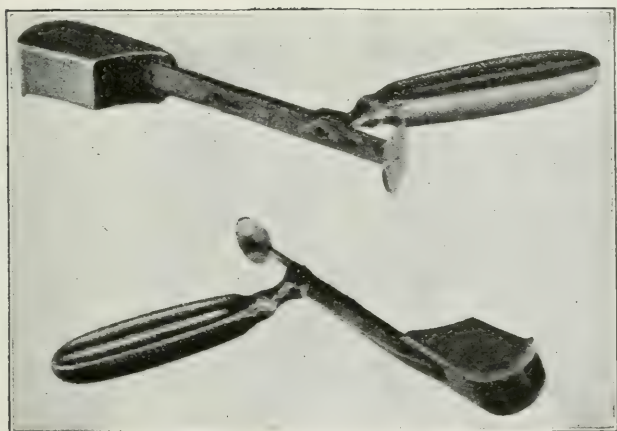


Fig. 2. Views of the direct vision adenotome.

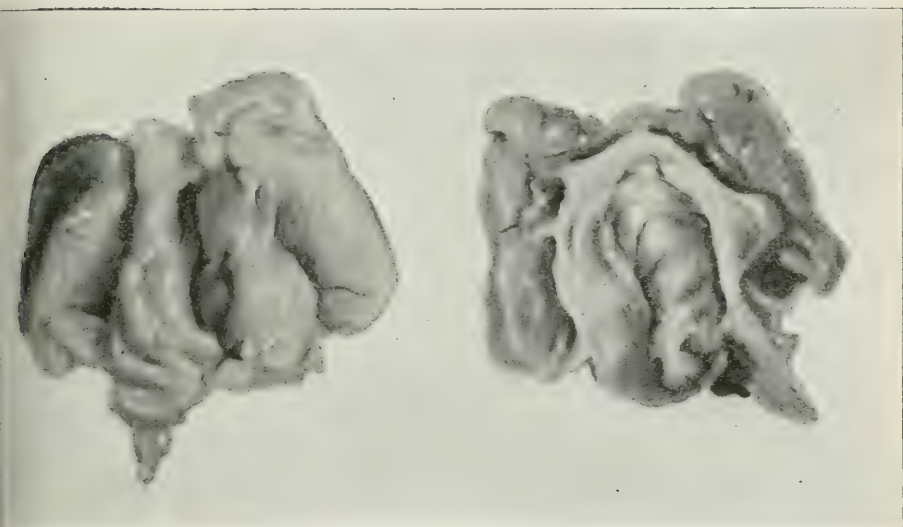


Fig. 3. Left view, anterior or presenting surface of an adenoid, showing crypts and elevations of lymphoid tissue; right view, posterior or capsular surface of adenoid, showing fibrous capsule with nodules and depressions similar to those found on capsular surface of the tonsil.

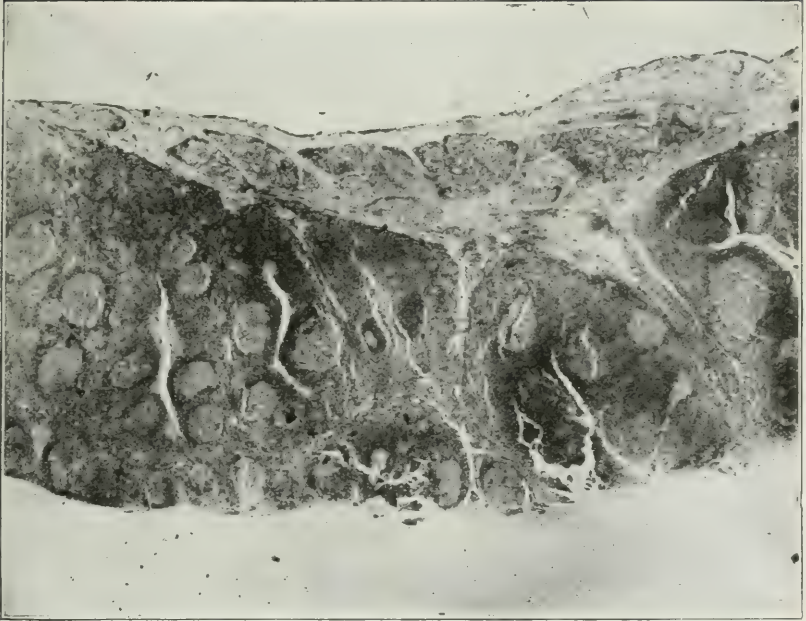


Fig. 4. Section through an adenoid, showing, above, a well developed capsule developed from the tunica propria of the mucous membrane containing mucous glands; fibrous trabeculae extend from the capsule toward the surface epithelium below; in the meshes of the reticular connective tissue are the leukocytes, among which are found numerous oval rings of lymphocytes or germinal centers.



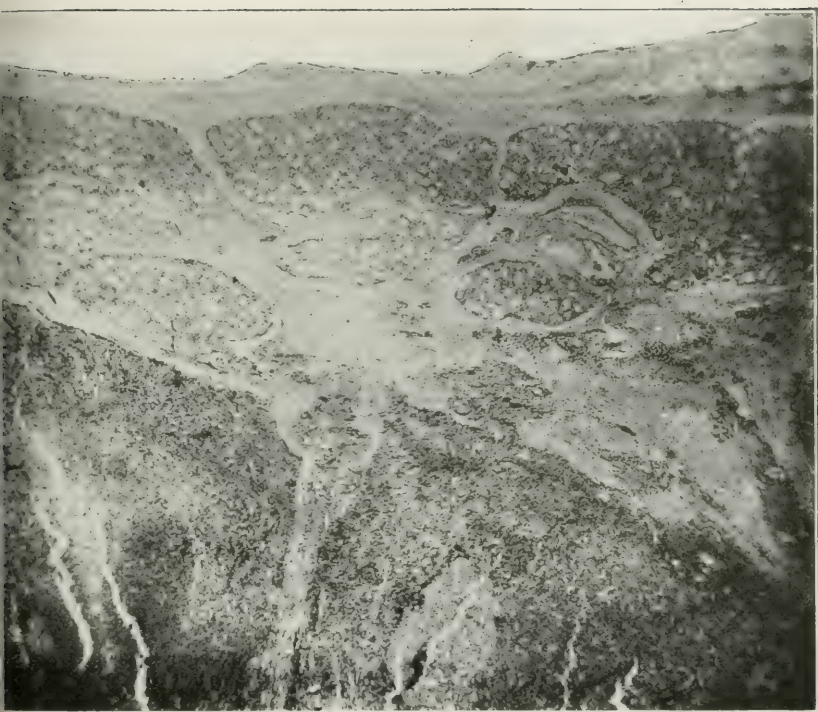


Fig. 5. Higher magnification of adenoid capsule. showing connective tissue of tunica propria, numerous mucous glands, and blood vessels and fibrous trabeculae from capsule descending into lymphoid tissue.

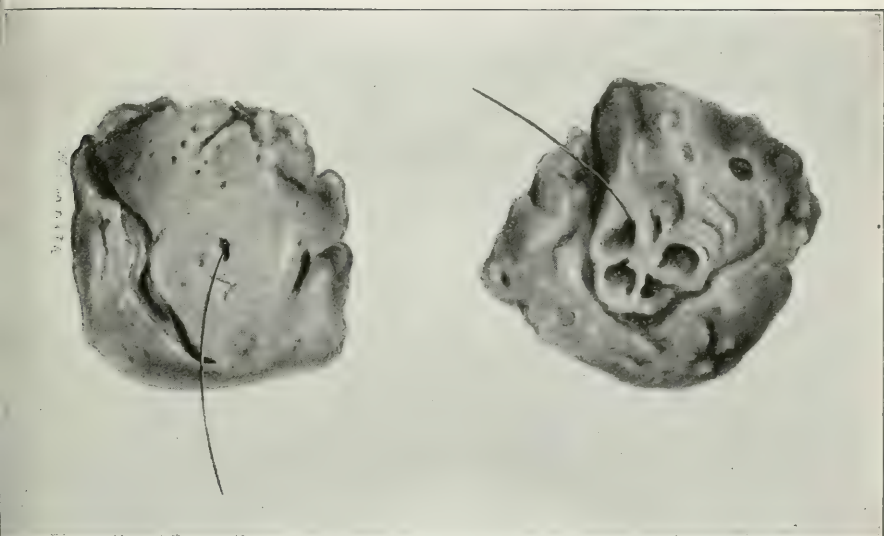


Fig. 6. Left view, anterior adenoid surface, showing opening with inserted bristle from which large quantities of pus and débris were seen to exude during removal; right view, posterior surface of adenoid, with capsule partially removed, showing multiple purulent abscesses occupying the body of the adenoid.

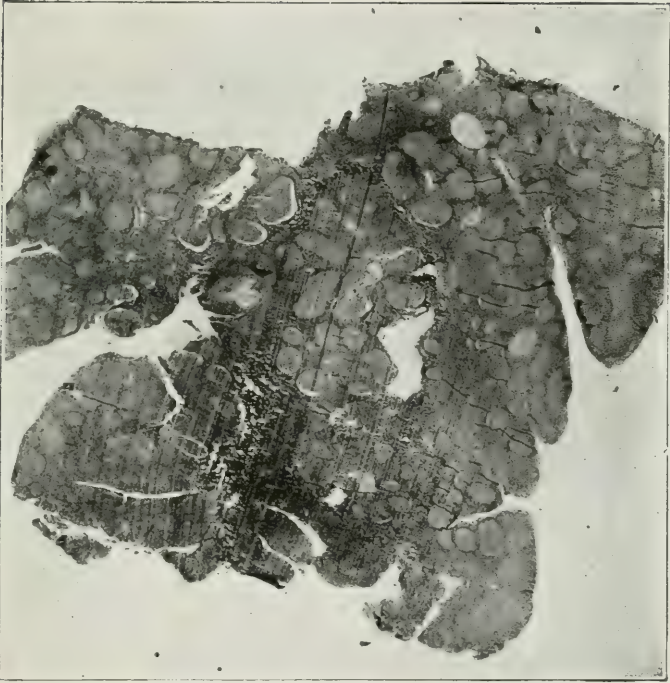


Fig. 7. Section through adenoid shown in Figure 6, with pus tract occupying center of largest adenoid mass.

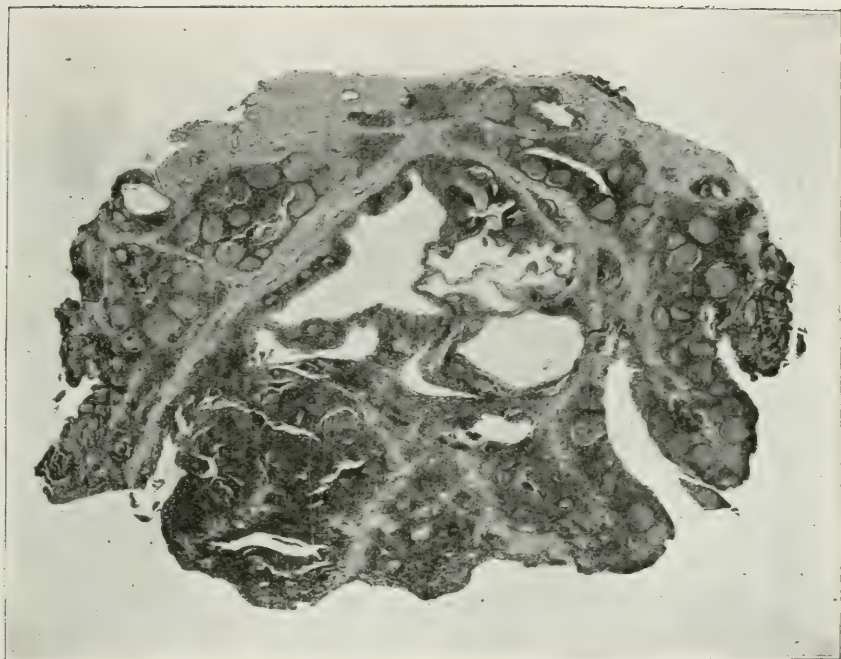


Fig. 8. Section through adenoid shown in Figure 7: Above is seen the adenoid capsule with trabeculae descending toward the epithelial surface, with the body of the adenoid occupied by large abscesses.

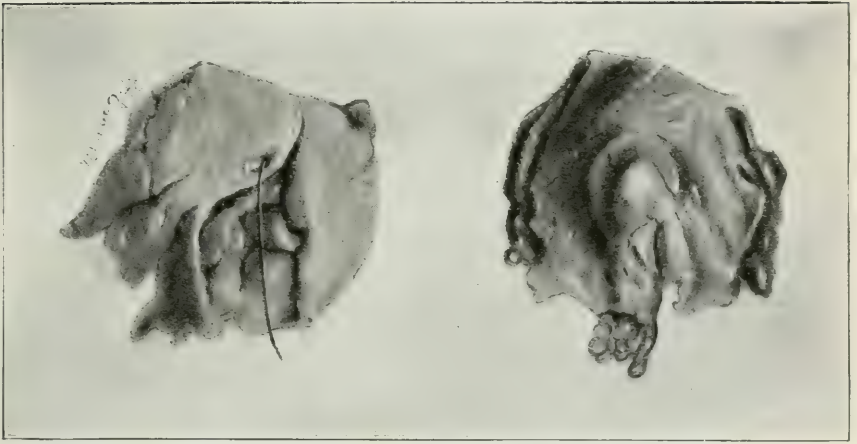


Fig. 9. Left view, opening containing a bristle on the presenting adenoid surface located to the side of the median bursa; right view, capsule of adenoid intact; the central elevated area was formed by a pus pocket seen through the capsule.



Fig. 10. Section from adenoid shown in Figure 9: A large necrotic area containing pus and destroying much of the lymphoid structure of the adenoid.

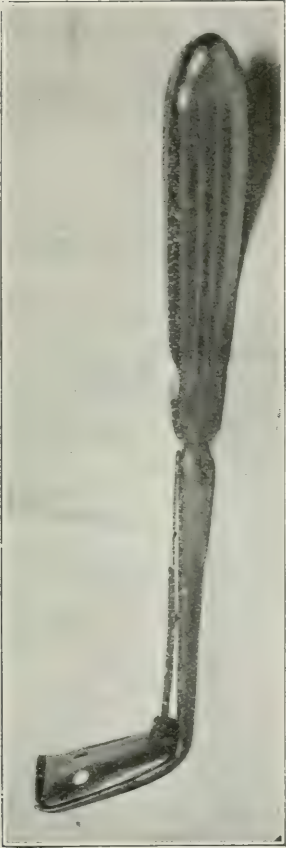


Fig. 11. Sluder's specially designed adenoid curet: A, closed;

LXXII.

A CASE OF EPITHELIOMA OF THE ESOPHAGUS WITH SOME UNUSUAL FEATURES— SPECIMEN.*

BY SAMUEL McCULLAGH, M. D.,

NEW YORK.

T. P., aged 57, consulted me on March 28, 1923, with the following history: About a year ago he first noticed a feeling as though there was an obstruction in his throat which interfered with swallowing. There was no pain, tenderness or interference with breathing. About nine months later he commenced to cough and raise thick tenacious mucus. About a month later blood appeared in the mucus in small amounts. This hemoptysis persisted for about a month, but was entirely eliminated by treatment. No history of hematemesis or regurgitation of food.

His principal complaint was the cough, which was exhausting on account of the tenacity of the discharge and the consequent difficulty in raising it. There was no pain on swallowing, though the difficulty persisted. On quiet breathing a distinct rattling was audible and marked dyspnea occurred on slight exertion. Has had a Wassermann test, which was negative. The sputum has been examined several times but no tubercle bacilli have been found. Has lost about 50 pounds in weight. An X-ray of the chest taken about a month ago was negative. Examination of the larynx showed a complete paralysis of the left cord, which was fixed, and a partial paralysis of the right cord which, on abduction, reached the cadaveric position. The whole larynx was congested but there was no evidence of new growth or ulceration. There was no marked adenopathy, though some enlarged glands could be felt below the larynx.

Dr. J. B. Solley, Jr., who examined his chest, reported: "The examination of the chest, on account of the stenosis, is

*Presented at the Forty-fifth Annual Congress of the A. L. A.

difficult. Percussion note is fair all over; there is no marked dullness or flatness. No rales. He coughed up a large 'gob' of thick yellow mucopus; there is therefore a raw surface somewhere in bronchi or alveolar spaces."

Dr. J. M. McKinney went over him from a neurologic standpoint and reported: "The neurologic examination, with the exception of the throat condition and a bilateral diminution of hearing, is negative. There is, apparently, a mass in the upper right mediastinum and some enlarged glands at the base of the neck. Pressure from this on the trachea might account for his breathing, but whether not it will explain the laryngeal paralysis I do not know."

Fluoroscopic examination with barium mixture showed some slight interference just above the clavicle, which apparently was confirmed by a plate, though this was very poor, and the interpretation was only made definite after the postmortem examination.

On April 6th, the patient's wife telephoned that he was choking to death, and he was ordered sent to the Manhattan Eye, Ear and Throat Hospital, where an emergency tracheotomy was performed by Dr. Robert Buckley. On opening the trachea two masses of tumor were coughed out, the larger piece being about 5 mm. by 3 mm., and the smaller about 3 mm. by 2 mm. A ragged tumor which bled very easily was seen on the posterior wall. The lumen of the trachea was so obstructed by this mass that Dr. Buckley inserted a piece of rubber tubing, as the tracheotomy tube that could be used was not long enough to go below the mass.

I saw the patient about an hour later, and though his condition had greatly improved, respiration was still much embarrassed. He was again taken to the operating room, where I removed the rubber tubing and inserted a metal tube, after establishing the lower limit of the tumor by means of a bronchoscope introduced through the tracheotomy wound. The patient sank rapidly and died the next day.

Permission for a postmortem was limited to enlarging the incision. Following is the report submitted by Dr. A. A. Eggston, pathologist at the Manhattan Eye, Ear and Throat Hospital:

"The body is that of a well developed but poorly nourished white man. In the anterior midline of the neck, below the thyroid cartilage is an incised tracheotomy wound.

"In dissecting the tissues of the lower neck and upper mediastinum a dense infiltrating tumor mass is encountered. This mass lies between the esophagus and trachea and extends laterally, filling the entire upper mediastinum. The bronchial and mediastinal lymph nodes are enlarged and very firm and involved by the tumor mass. The esophagus, trachea and tumor mass is removed in toto.

"Upon opening the esophagus the entire upper one-third is indurated and shows an ulcerated area 10 by 6 mm. and covered with friable masses. In the lower part of the esophagus there are several tumor metastases upon the mucosa.

"Upon opening the trachea there is found a sessile oval tumor 4 by 2 cm., attached to the posterior wall. This mass is friable and hemorrhagic. The mass is 3 cm. above the bifurcation of the trachea. The tumor mass, between the esophagus and trachea, is very firm and measures 11 by 5 cm. The mediastinal nodes are likewise indurated.

"Microscopic examination of the esophagus and tracheal tumors and of the tumor proper show irregular areas and masses of rather large epithelial cells. The cells form no normal histologic structures and many show active mitosis. Many areas of necrosis are present in the epithelial cells. These sections are similar to the ones made from the tissue which was expelled by coughing before death.

"Diagnosis: Medullary carcinoma."

The points of particular interest to me are: (1) The friability of the tumor, the detachment of at least one of the fragments expelled at the time of the tracheotomy undoubtedly having been responsible for the sudden attack of asphyxia, and (2) the slight subjective symptoms until just prior to the end.

LXXIII.

SURGICAL DIATHERMY BY THE NEEDLE METHOD.*

BY ALFRED LEWY, M. D.,

CHICAGO.

Diathermy, thermopenetration or electrocoagulation, as it has been variously named, depends upon the heating of the tissues by their resistance to the electric current. For this purpose the high frequency (modification of the D'Arsonval¹ current is used, the interruptions, or rather alterations, being so frequent that they do not arouse nerve impulse or muscular contractions except when through poor contact sparking takes place, or the frequency is lowered otherwise. For surgical purposes a large indifferent electrode and a small one, or a needle inserted into the tissues, is used, so that where the current is concentrated around the small electrode intense heat is generated, causing rapid coagulation of the tissues—in fact cooking them. A number of works,^{1 2 3 4} describing various appliances, operating technic for different conditions, etc., have been published, so it is my intention to describe only a few additional observations of my own.

This subject seems first to have attracted attention to any extent as a therapeutic resource about 1909, when articles by Von Berndt⁵ and Laqueur⁶ appeared, followed shortly by Nagelschmidt,⁷ who has since done a great deal of work on the development of apparatus and its clinical application, and probably introduced it to the British⁸ in 1910, also writing a textbook (*Lehrbuch*) on the subject in 1913. Its surgical application, particularly in malignant growths, was thoroughly worked out by Doyen,⁹ who probably deserves the greatest credit for work in this connection, although Cumberbatch¹⁰ reports cases treated as far back as 1910.¹¹ Many others appeared in increasing numbers in foreign literature. In our country Clark, who refers to it as "dessication,"¹²

*Candidate's thesis. American Laryngological, Rhinological and Otological Society.

Corbus,¹³ DeKraft,¹⁴ Kolischer and Eisenstaedt¹⁵ and Plank,¹⁶ have done considerable work with this procedure in various parts of the body, and Novak¹⁷ especially in the larynx.

It was from Dr. T. Howard Plank that I learned the technic which I use, namely, the needle in nearly all cases, because, as I believe, of its wider and more accurate application, the reduction in the number of electrodes, and the possibility of developing a more uniform technic.

The first essential is a machine which will deliver a current of just sufficient voltage to insure its traversing the entire body, or as much thereof as lies between the two electrodes, and not so high that there is great difficulty in insulating the cords and electrodes with which we have to work. The amperage must be sufficient to generate enough heat at the small electrode to coagulate the tissues immediately surrounding it. In working in the mouth and throat 500 to 1,000 milliamperes are usually sufficient, although I have used higher, and higher amperages are required for larger masses of tissue. The modern machines, of which there are a number on the market, are capable of fairly smooth control from small amounts of current for diminutive lesions, for instance, about the eye, up to five or more amperes. In this we have quite an advantage over the men who had to work with less flexible and convenient apparatuses, and who still achieved some excellent results. A good foot switch, under immediate control of the operator, and well insulated cords and electrodes to prevent unexpected shocks to the operator and undesirable burns to normal tissues, are indispensable. Most men use a large electrode, at least eight by six inches, covered by towels or some special tissue, wet with 5 to 10 per cent salt solution. This must be kept uniformly wet, as if it dries out in places, all current passes through the remaining wet area, and may be sufficiently concentrated to cause a burn. As the electrode dries out rapidly under the heat generated by the current, this is an important thing to watch. By using an uncovered block tin electrode, carefully applied to the body, having it bandaged on and the patient lying on it, the connecting wire hooked into an eyelet, this part carefully protected, much of the danger of a burn can be obviated. The operating needle, which should be in a well insulated handle, may be of any

desired length with sufficient strength, as it can be covered with a rubber catheter, exposing only so much as the depth we wish to penetrate in order to cause the right amount of coagulation. Various metals have been used, but Dr. Emil H. Grubbe has suggested to me iridioplatinum (commonly used with the galvanic current) as best fulfilling the indications. Other operators seem to prefer disc, button or ball electrodes, and in some cases a knife for excising and coagulating at the same time. These are all described in the textbooks mentioned above. The needle is inserted before switching on the current, which is again turned off before removing the needle, otherwise sparking takes place, causing muscular contraction or a burn where it is not wanted. This sparking, however, can be turned to good use in stopping bleeding by charring the surface.

The special advantages of the needle are: That it does away with a considerable number of special tips; that they can be placed accurately, but particularly that it can be made to traverse several layers of tissue, through fascial planes, with a greater probability of accuracy in the amount and depth of tissue coagulated. This is well shown in figure 1, photograph of a piece of meat, in which four punctures were made. Each one was given twenty seconds' application of 700 milliamperes. The similarity of all except one will be noted, which shows coagulation extending at an angle from the tip. This, I believe, was caused by tilting the needle in situ, separating the muscle bundles and permitting the steam generated in the moist tissues to travel along the separation. I shall refer to this steam production further on.

Figures 2 and 3 show a piece of meat composed of two muscle layers, separated by fascia, which were treated by a disc electrode (the method mostly used by others). Figure 2 shows the surface, figure 3 a cut through one of the coagulated areas. Figure 4 the same, enlarged three diameters. The applications were also twenty seconds each, of 800 milliamperes. In these pieces the surface coagulation is wider, but the depth is not so great. The picture does not show it clearly but it does not extend quite down to the fascia, although the total thickness of the meat was slightly over one inch, as against two inches. If we can draw any conclusions from

these two experiments, it would seem that the disc electrode would be useful for more superficial work in homogeneous tissue, while the needle should be preferred for deeper application and where more than one layer of tissue is to be coagulated. The needle can take the place of the surface electrode by making a shallow insertion.

Most writers advise that the application be stopped as soon as steaming is observed, so as to avoid sparking, which occurs as soon as an area becomes carbonized, and thereby a poor conductor, whereupon the heating lessens, and theoretically the coagulation in the deeper parts ceases. While this is relatively so, I do not think it is so important when using the needle, as I have observed the coagulated area to extend even after the needle track is carbonized and sparking begins. The question of steam production in the tissues is, I believe, an important one, and one that does not seem to have received sufficient attention. It is difficult to calculate along what planes the steam will travel, and the steam, of course, cooks the tissues as well as the heat from resistance to the current. A detailed knowledge of the anatomy of the parts may be of help, and undoubtedly we will learn from experience. While the circulation of the blood in living tissue tends to dissipate the heat, the presence of greater moisture permits the coagulation process to become wider before carbonization, and this fact is taken advantage of by some,¹⁸ who drop salt solution on the part on which they are working. It also leads to greater steam production, which, as pointed out above, is one of the factors which prevents accuracy in localizing the destruction of tissue, as desired.

The proximity of the large vessels in the neck, and of important nerves, particularly the pneumogastric, must always be kept in mind in working in this region. To some extent the blood vessels are protected by the rapid flow of blood through them, acting much as does the water in an automobile radiation system in dissipating the heat. In cases 1 and 2, described by me, I inserted the needle behind the tonsil as is done in block anesthesia for tonsillectomy. I realized that I was taking chances, but believed it was justified by the almost certain fatal termination, if the growth was not entirely destroyed. Fortunately there was no secondary hemorrhage,

although this has occurred in some cases. Preliminary ligation, especially of the lingual, has been advised. I am not sure as to the advisability of this in the case of the carotid and jugular, as their circulation probably also protects to some extent the pneumogastric nerve. If a malignant growth has penetrated to this depth it is probably hopeless except for temporary relief at any rate. Because of the outpouring of fluid into the tissue adjacent to the coagulated area, operations in or about the larynx require tracheotomy. Where dessication (fulguration) alone is used, this will probably not be necessary. I am using the term dessication in preference to fulguration, because dessication describes what takes place in the tissue when the spark is used. This process is useful for superficial or diminutive lesions, and has the advantage in skin lesions of leaving a smooth white scar, where the coagulation caused by actual contact of the electrode may cause keloid.

The advantages and disadvantages of this method of surgery are well summed up by Saberton:¹⁹

1. Tumors otherwise inoperable may be attacked.
2. The operation is more or less bloodless.
3. The danger of metastasis is much less than with a cutting operation, owing to the sealing of the blood vessels and lymphatics draining the parts.
4. Sterilization of the parts.
5. There is complete destruction of the visible and palpable malignant disease.
6. The operation is rapid and easy.
7. There is no surgical shock.
8. Convalescence is rapid.
9. Formation of adhesions is rare.
10. The operation can be repeated if necessary.

Disadvantages:

1. Healthy and diseased tissues are equally destroyed.
2. The surgeon cannot see important vessels and nerves.
3. There is danger of secondary hemorrhage when operating near large blood vessels.
4. There is a tendency to formation of keloid in operations involving skin surfaces.

To this last I may add that in operating near bone, destruction of the periosteum may lead to long continued suppuration.

To No. 7, I may add that while surgical shock may usually be negligible, the necessity of using chloroform as an anesthetic increases the anesthetic risk. Ether can be used by first giving morphin-scopolamin enough to have the patient quite stuporous, then using just enough ether to complete anesthesia, and before beginning the diathermia removing the ether from the room. I have heard of one case of ether explosion caused by this method, and in one of my cases prolonged cyanosis, supposedly due to the heavy dosage of morphin and hyoscin, caused a great deal of anxiety. The disadvantage No. 1, "Healthy and diseased tissues are equally destroyed," is not absolutely accurate, as Doyen has shown²⁰ that the malignant cells succumb to a somewhat lesser degree of heat than do mature cells.

I believe that diathermy presents distinct advantages over any other method for removal of malignant growths in the mouth and throat, and for that matter in several other regions, and have therefore taken the liberty of adding my few observations to the fundamental work already done by others. The removal of malignant growths by this method, as with other methods, should be supplemented by subsequent raying, by removal of gland bearing tissue where necessary, and by the use of such measures and remedies as experience has apparently proven useful. With potassium nitrate intravenously, claimed by some to relieve the pain of inoperable cancer, I have had but one experience, and that not favorable. Arsenic in tonic doses I have used a number of times, but cannot yet present any definite data concerning it.

Case 1.—Male, age 55. For over two months complains of severe pain in right side of throat. Swallowing difficult; has lost considerable weight and strength; looks cachectic. Examination shows ulcerative process involving right tonsil, both pillars in part and the adjacent part of the tongue; induration extends toward angle of jaw; no perceptible cervical adenitis. Section removed shows squamous celled epithelioma. Operation: Electrocoagulation (diathermia) by the needle method, destroying all the involved tissue. Patient was free from pain immediately following operation, and ate in comfort for the first time in months. Most of slough separated in two weeks, but healing was delayed by a splinter of bone from ramus of

mandible which had evidently been overheated and which was extruded in three weeks. This patient had no dissection of glands of neck and no raying (this was four years ago). He remained well, but nine months later a swelling appeared in infraclavicular region which eventually resulted in death. There was no recurrence in situ.

Case 2.—Male, age 53. For three months has had pain in right side of throat and tongue. Examination reveals ulcerative process on the right side of the tongue, right tonsil and both pillars, with quite extensive induration toward base of tongue and into floor of mouth. Section made at time of operation showed squamous celled epithelioma. Attempt was made to coagulate the entire area, and block dissection of neck followed, but healing did not take place, and there was further invasion of contiguous tissues. Another but unsuccessful effort was made to destroy the invaded area. This patient had no relief, even temporary, from the operations and eventually died.

Case 3.—Male, age 66. Has had pain in throat and up into head for three months. Examination shows an ulcerative process involving left tonsil, both pillars, soft palate and posterior pillar of right side. There was very little induration, and smears showed fusiform bacilli and spirilli in profusion. He was treated for a brief period for Vincent's angina, then section was made, which showed the process to be a squamous celled epithelioma. Two months were lost in radium applications, under which the process progressed. With diathermy the entire involved area was coagulated, including soft palate, and, after the slough separated, the original area looked well, but there was no relief of pain, which was severe, and shortly there appeared new nodular areas in the nasopharynx, which, because of the absence of the soft palate, were easily reached for another attempt at coagulation. There was no relief and soon after the second operation the patient went into stupor and died two weeks later. In this case I believe the brain was invaded, as no urinary or other findings explained the stupor.

Case 4.—Male, for two months has had pain on swallowing, located on right side of tongue. Has lost weight and strength, probably largely from lack of nourishment, as, although he

looked badly, there was no definite cachexia. Examination showed deep fissure in right side and dorsum of tongue, opposite the tonsil, with two fungating nodules also on dorsum. The posterior pillar appeared to be slightly infiltrated. Section taken at time of operation showed the process to be a squamous celled epithelioma. The posterior pillar, part of tonsil and an area of tongue extending beyond all the involved area were coagulated. There was no relief of pain until the slough separated, about two weeks later. This operation was followed by block dissection of the right side of neck and a course of X-ray. Following the first X-ray treatment there was a large infiltration of the tissues of the neck, which entirely disappeared in three months. This patient was also put on a colloid preparation of arsenic in tonic doses for nearly a year. It is now (October, 1922) over two years since the operation and the patient is well, has gained in weight and strength and, since three months after the operation, has been working as usual. The tongue was quite stiff for a while, but is now more movable. It can be protruded just beyond the teeth. There has been an annoying paresthesia of the right side of the tongue, which still persists, and about the right one-third is also anesthetic. The defect in the side of the tongue left by the operation is plainly seen.

NOTE.—Patient No. 4 is still living and well.

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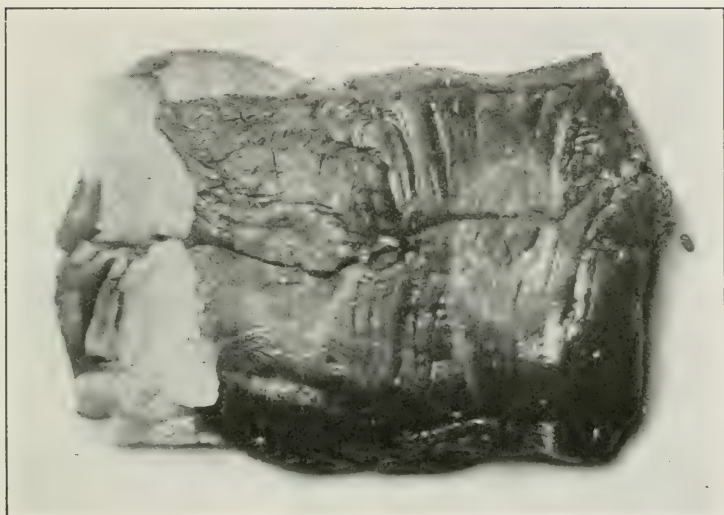
110 N. WABASH AVE.



—Photo by Dr. J. W. Lowell.

FIGURE 1.

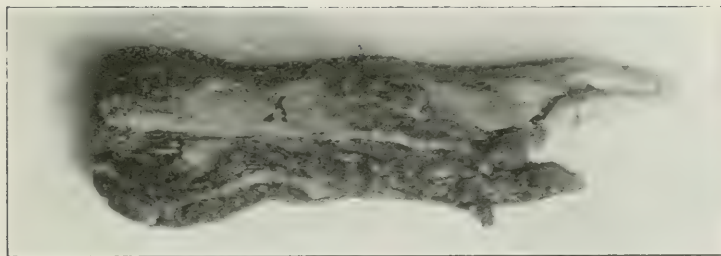
Piece of meat, two inches thick; four punctures, 20 second applications of needle at 700 milliamperes. Cut through punctures and laid open. Notice how coagulation tends to follow the "grain" of the fibre bundles; probably due to steam. Otherwise the extent of coagulation is quite uniform.



—Photo by Dr. J. W. Lowell.

FIGURE 2.

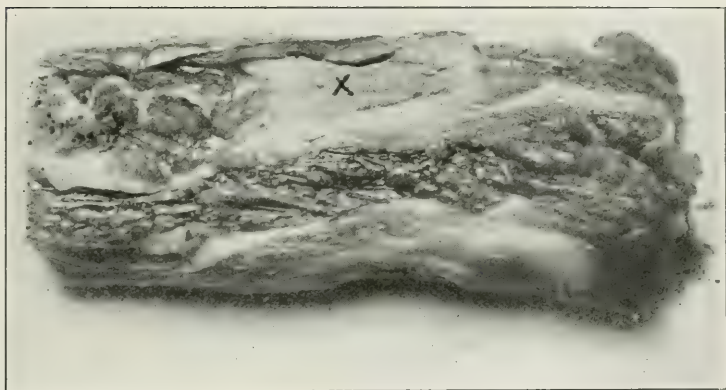
Showing size of surface coagulation with flat electrode 6 mm. diameter, 20 seconds' application, 800 milliamperes.



—Photo by Dr. J. W. Lowell.

FIGURE 3.

Same piece as Fig. 2, on cut section. The coagulated area, marked X, appears to reach the fascia, which in reality it did not quite do. The surface at the point of section is at the top of the coagulated area. The meat behind it is pushed up a little higher, and appears higher on the picture.



—Photo by Dr. J. W. Lowell.

FIGURE 4.

The same as Fig. 3, magnified about two diameters. To the left of the coagulated area is fat, and below it is fascia showing white. The coagulated area appears gray.

LXXIV.

FURTHER STUDIES OF FATALITIES FOLLOWING OPERATIONS ON THE NOSE AND THROAT.*

BY H. W. LOEB, M. D.,

ST. LOUIS.

In a paper read before this Society a year ago, I presented a study of 332 hitherto unreported fatalities following operations upon the nose and throat.

Since that time I have collected 112 more, making a total of 444 cases which constitute the basis of this paper. How many more there are which have thus far eluded this inquiry is a matter of conjecture, though it is likely that the number is not inconsiderable.

It has been my experience that, whenever this subject is brought up at medical conventions or privately among several laryngologists, quite a number of unincluded cases have been added.

A summary of the grouping of the cases, according to the previous and present reports, yields the following:

	Previous	Additional	Total
1. Meningitis	124	44	168
2. Hemorrhage	55	21	76
3. General sepsis	20	7	27
4. Erysipelas	8	1	9
5. Endocranium (excluding meningitis).....	27	4	31
6. Respiratory tract	43	12	55
7. Heart	5	0	5
8. Miscellaneous	20	16	36
9. Undetermined	30	7	37
Total	332	112	444

It is to be noted that there has been no material change in the predominance of one type of fatality over another. However, there has been an increase of 35 per cent in the reports of meningitis and 40 per cent in those of hemorrhage, although the total number of cases added is only 30 per cent. As in the previous report, care has been observed to exclude

*Read before the meeting of the American Laryngological, Rhinological and Otological Society, May, 1923.

cases, the history of which was incomplete, doubtful or inexplicit.

I. MENINGITIS.		Previous	Additional	Total
1. Tonsillectomy	4	3	7	
2. Intranasal frontal operation.....	16	3	19	
3. Probing and irrigating frontal sinus.....	7	0	7	
4. Ethmoid operation, with or without resection of the middle turbinate.....	39	23	62	
5. Sphenoid operation	10	0	10	
6. Maxillary operation	1	1	2	
7. Resection of middle turbinates.....	15	5	20	
8. Submucous resection	13	2	15	
9. Removal of polypi.....	13	2	15	
10. Combined sinus operation.....	1	3	4	
11. Combined submucous and sinus operation	5	2	7	
Total	124	44	168	

Three cases of meningitis following tonsillectomy have been added to the list. Of the 41 others, 23 are ascribed to ethmoid operations, making a total of 62, and 18 fatalities were due to operations including or adjacent to the ethmoid cells.

II. HEMORRHAGE.		Previous	Additional	Total
1. Tonsillectomy (with or without coincident adenoidectomy	43	19	62	
2. Adenoidectomy alone	2	0	2	
3. Incision of peritonsillar abscess	3	0	3	
4. Submucous resection	2	0	2	
5. Sinus operation	4	2	6	
6. Resection inferior turbinate.....	1	0	1	
Total	55	21	76	

The increase has been almost exclusively due to tonsil operations.

III. GENERAL SEPSIS.		Previous	Additional	Total
1. Intranasal maxillary operation.....	2	0	2	
2. Submucous resection	1	2	3	
3. Tonsillectomy	16	3	19	
4. Incision peritonsillar abscess.....	1	1	2	
5. Probing frontal sinus.....	0	1	1	
Total	20	7	27	

IV. ERYSIPELAS.		Previous	Additional	Total
1. Submucous resection	4	0	4	
2. Sinus operations	2	0	2	
3. Resection middle turbinate.....	2	0	2	
4. Tonsillectomy	0	1	1	
Total	8	1	9	

The addition of a fatality due to tonsillectomy is to be noted.

V. ENDOCRANIUM, EXCLUDING MENINGITIS.

	Previous	Additional	Total
1. Brain abscess	5	1	6
2. Cavernous sinus thrombosis.....	9	3	12
3. Cerebral embolus or hemorrhage.....	13	0	13
Total	27	4	31

Three new cases of cavernous sinus thrombosis make the possibility of this complication a matter of some concern.

VI. RESPIRATORY TRACT.

	Previous	Additional	Total
1. Foreign body	5	0	5
2. Edema larynx and pharynx.....	6	0	6
3. Pulmonary edema	1	0	1
4. Pneumonia	17	5	22
5. Pulmonary embolus and abscess.....	14	7	21
Total	43	12	55

Reports of pulmonary abscess have been increased 50 per cent. We may be sure that there are many yet to come.

VII. HEART.

	Previous	Additional	Total
1. Angina pectoris	1	0	1
2. Dilatation	1	0	1
3. Asthenia	1	0	1
4. Infarct	1	0	1
5. Rupture	1	0	1
Total	5	0	5

VIII. MISCELLANEOUS.

	Previous	Additional	Total
1. Acidosis	1	0	1
2. Delirium tremens	1	0	1
3. Dilatation of the stomach.....	1	0	1
4. Diphtheria	1	1	2
5. Epilepsy	1	0	1
6. Hyperthyroidism	2	0	2
7. Orbit injury	1	0	1
8. Poisoning	1	0	1
9. Scarlet fever	3	0	3
10. Shock	1	3	4
11. Status lymphaticus	5	7	12
12. Uremic coma	1	3	4
13. Uterine hemorrhage	1	0	1
14. Convulsions	0	1	1
15. Acute lymphatic leukemia.....	0	1	1
Total	20	16	36

IX. UNDETERMINED.

	Previous	Additional	Total
1. Tonsillectomy	16	4	20
2. Adenectomy alone	1	0	1
3. Exenteration ethmoid, with or without removal of polypi.....	4	1	5
4. Puncture of nasomaxillary wall, irrigation of the sinus.....	5	1	6
5. Submucous	1	0	1
6. Resection middle turbinate.....	2	0	2
7. Combined tonsillectomy and sub-mucous resection	1	0	1
8. Incision peritonsillar abscess.....	0	1	1
Total	30	7	37

Many of the cases reported carry with them important lessons of indication and contraindication, of inexpertness, of danger of multiple operations, of postoperative inattention and of unforeseen accident.

The following may be cited:

1. Incision of a supposed peritonsillar abscess followed by death from acute lymphatic leukemia.

2. Another from myelogenous leukemia after a tonsillectomy.

3. Acute nephritis following combined submucous resection and tonsillectomy in a patient suffering from nephrolithiasis.

4. Septicemia and phlegmon of the neck following combined submucous resection and tonsillectomy.

5. Tonsillectomy in a patient with hydrocephalus followed by meningitis.

6. Meningitis following resection of the middle turbinate: patient who left town on the same day, having a hemorrhage that night, had his nose plugged by inexpert physician.

7. Extensive removal of polypi followed by meningitis, autopsy showing that a portion of the cribriform had been torn away.

8. Cavernous sinus thrombosis following complete exenteration of both ethmoid labyrinths and resection of the anterior wall of both sphenoids.

9. Tonsillectomy in a patient who was intoxicated one day before the operation and four days after. Death from meningitis.

10. Three days after an acute cold patient developed severe pain in the face. Diagnosis pansinusitis. All the sinuses were opened intranasally on the same day. Patient conscious with a temperature of 102 degrees. After operation, patient lost consciousness and became comatose with a temperature of 106,^o profuse discharge from the nose and symptoms of meningitis. Radical operation on sinuses on both sides performed but patient promptly died of meningitis.

11. Acute frontal sinusitis, anterior end of middle turbinate removed for drainage on account of pain. On the third day the patient died from meningitis.

12. Polypi removed with biting forceps, both middle turbinates resected and ethmoid cells, both sides, exenterated. Operator was able to see pulsation in region of cribriform plate with escape of cerebrospinal fluid. Death from meningitis in three days.

13. Fatality from meningitis on the third day after exenteration of both ethmoid labyrinths with Ballenger's knife.

14. Tonsillectomy in a patient who had had two attacks of Vincent's angina. Several days later cervical lymphatics became swollen and tender, with a temperature of 107 degrees. Incision resulted in the evacuation of a small amount of very offensive pus which contained fusiform bacilli and Vincent's spirilla. Patient recovered rapidly but ten days later developed pulmonary abscess from which he died.

15. Fatality from acute uremia following a local tonsillectomy in a marked nephritic.

16. Fatal meningitis following pansinus operation three days after onset of attack.

17. Tonsillectomy followed by death in 12 hours from hemorrhage in a hemophiliac.

18. Injury to the meninges in removing polypi, brain substance coming through the nose while the patient was lying in bed. Confirmed by autopsy.

The study of these cases has served to strengthen the views expressed in my previous paper and impels me to urge all who meet with these unfortunate experiences to insist upon autopsies and complete autopsy protocols, to the end that we may have a better understanding of the processes involved and better means of combating them.

LXXV.

THE ANATOMY OF THE SPHENOID FISSURE.*

BY GREENFIELD SLUDER, M. D.,

ST. LOUIS.

In 1914† I injected the nasal (sphenopalatine, Meckel's) ganglion and paralyzed the abducens nerve. The abducens palsy recovered in three months. All diplopia vanished. I accepted this as the logical sequence of such an accident according to the accepted anatomic understandings of the anatomy of the sphenoid fissure—i. e., that the alcohol passed upward and outward from the pterygomaxillary fossa and reached the sphenoid fissure, meeting there the lowermost nerve—the abducens. The tissues were very loose in this case. The alcohol met with no resistance.

Five years ago Dr. Joseph C. Beck injected the nasal ganglion and paralyzed the oculomotor nerve, in a patient who later consulted me. This led me to think that the anatomy of the sphenoid fissure was subject to variations.

The accompanying drawings of dissections of the sphenoid fissure show that the positions of the nerves are not constant. The abducens is probably oftenest the lowermost. Sometimes, however, the oculomotor, if not in its full trunk certainly in some of its branches, is lowermost, which accounts for the clinical phenomena just mentioned.

*Presented to the American Laryngological Association, May 18, 1923.

†Ewing, A. E., and Sluder, Greenfield: Abducens Palsy following Nasal Trauma and Nasal Infection, *Am. Jour. Ophthal.*, December, 1914.

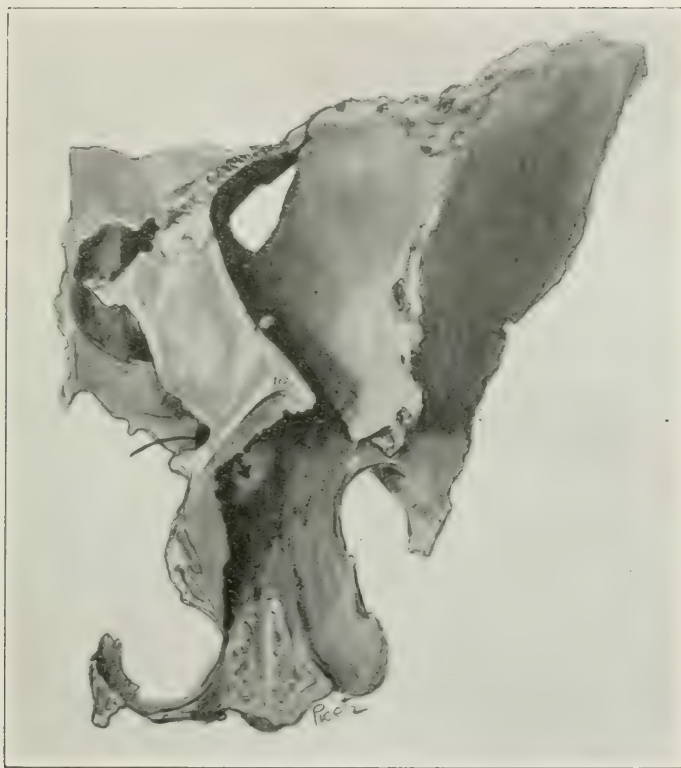


FIGURE 1.

Bony environments of sphenopalatine foramen and sphenoid fissure, demonstrating their relations and the distance between them. The foramen rotundum also appears.

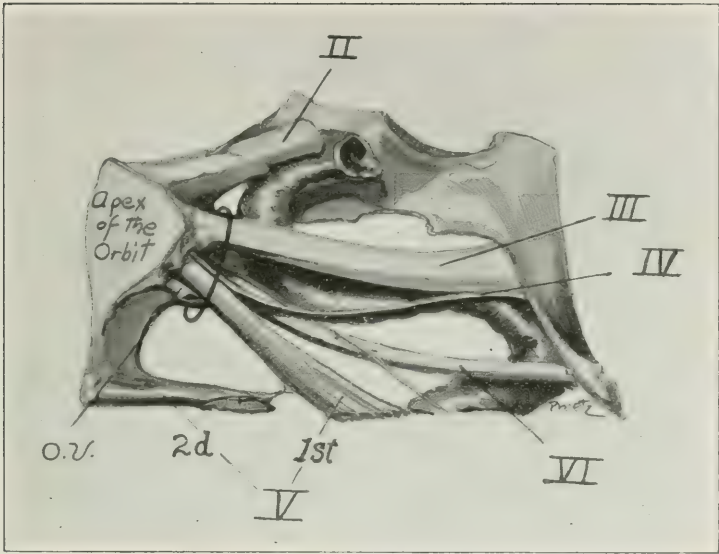


FIGURE 2.

Semidiagrammatic representation of the contents of the sphenoid fissure, showing their relations to one another, both in the fissure and in the cavernous sinus. Left side. The sixth nerve as it passes through the sphenoid fissure here lies mesial to the fourth and the first division of the fifth above the ophthalmic vein. Note the position of the internal carotid with relation to the other structures.

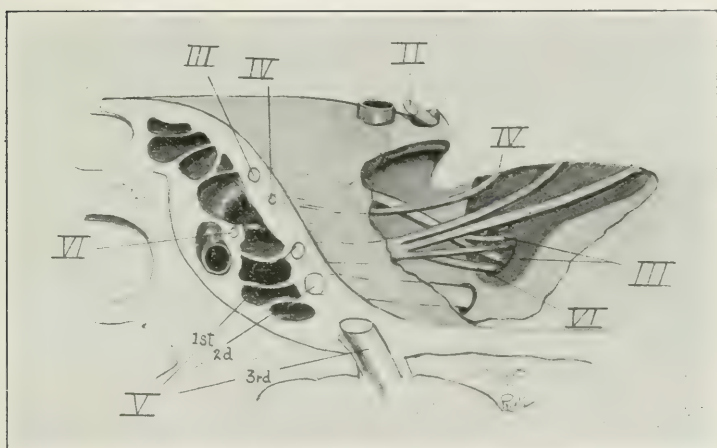


FIGURE 3.

Relations of the structures in the cavernous sinus and the sphenoid fissure as depicted in Cunningham's Anatomy.

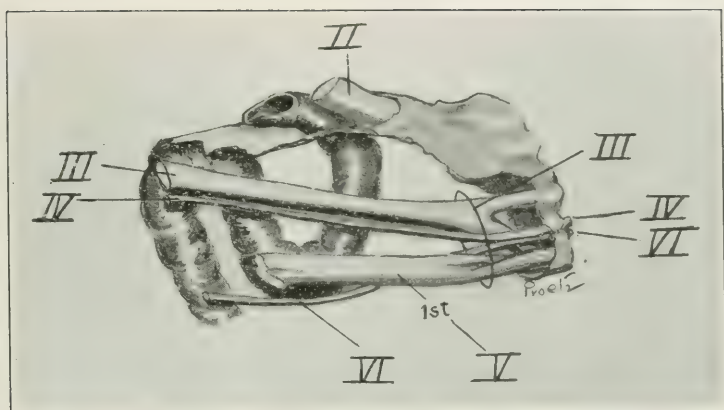


FIGURE 4.

Contents of the sphenoid fissure right side. In this case the sixth nerve passing through the cavernous sinus mesial to, and at a lower level than the first division of the fifth, ascends still mesial to it and traverses the sphenoid fissure above it and in conjunction with the fourth. Note the variation in plane and degree of tortuosity of the internal carotid as compared with Fig. 2.

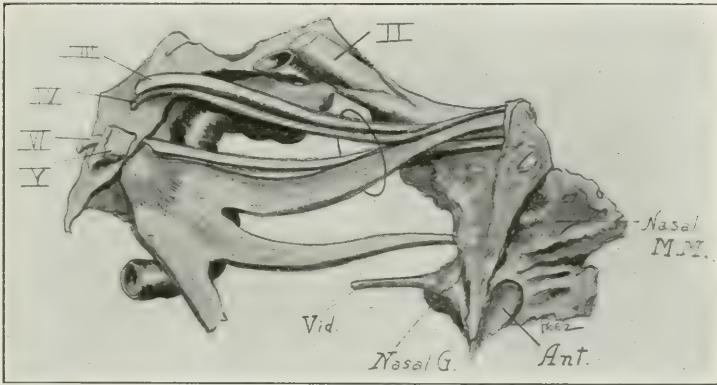


FIGURE 5.

The contents of the sphenoid fissure, showing their relations to one another and to the nasal ganglion and the vidian nerve. The intimate relation of the ganglion to the posterior superior extremity of the maxillary sinus is also shown.

Lateral view of structures on the right side.

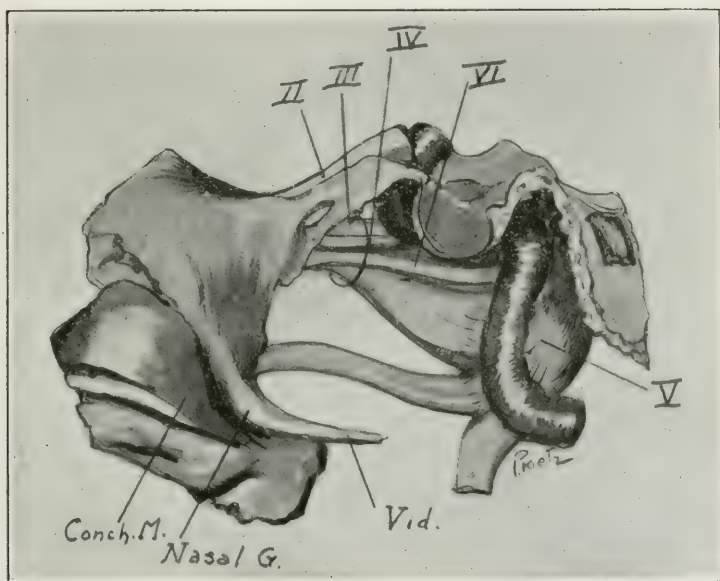


FIGURE 6.

The same dissection shown in Fig. 5, but viewed from the mesial side. The relations of the concha medialis, the nasal ganglion and the contents of the sphenoid fissure are shown.

LXXVI.

DEAFNESS AS A SEQUELA TO MUMPS.*

BY GEORGE H. WILLCUTT, M. D.,

SAN FRANCISCO.

A number of articles have appeared on the subject of deafness as a complication or sequelæ of mumps during the last five years, the most instructive of same having been presented by Stern, Radin, Heilskov, Kaunitz and Albright. In the year 1860, Toynbee reported the first case of deafness associated with this disease and then followed reports of a number of other cases during a period of years from 1880 to 1890. In the year 1885, Pierce reported in the Manchester Chronicle an interesting series of forty cases which he had collected. However, it has remained for the past thirty or forty years a most unsettled pathologic question as to what is the anatomic cause of this severe, afebrile, incurable disease of the auditory nerve, which arises during or following acute epidemic parotitis.

To briefly summarize the various pathologic divisions or classes in which the reported cases have been placed, there are three main divisions: (1) those in which only the cochlear branch of the auditory nerve is affected, (2) those in which the vestibular branch is the only affected part, and (3) those in which both cochlear and vestibular branches are involved.

Varying theories of the mode of infection of the auditory nerve have also been expounded by the various authors, such as (1) direct extension, (2) direct transplantation, (3) by way of nerve sheaths or blood vessels, (4) by an acute exudation or metastasis in the labyrinth, and (5) a neuritis of meningitic origin. The authors of the last few years mentioned above have divided about as follows: Heilskov and Kaunitz favor the neuritis of meningitic origin, Albright and Weinstein and others, that of metastasis or exudative labyrinthitis. In brief, the favored theories are one or the other of the follow-

*Candidate's thesis accepted by the American Laryngological, Rhinological and Otological Society.

ing: a neuritis descending from the meninges toward the labyrinth, or a neuritis ascending from the labyrinth toward the brain center.

With these two theories before him, the author of this work has attempted to add something to the literature which may prove of some benefit in the future work on the subject and aid in clearing up the differences of opinion now existing. It is with this hope that the following cases are reported in detail and the suggestions offered for further investigations.

Since June 26, 1916, five cases of complete deafness following or during an attack of acute epidemic parotitis have been under the author's observation. In this series of cases, three were in children between the ages of five and eleven years, and two were female adults, aged 25 and 35 years.

Case 1.—In June, 1916, a woman telephone operator, 29 years of age, was referred with the following history: On May 27th, she had contracted mumps from her young daughter, while nursing her through an acute attack of the disease, and had been herself under the care of her family physician. On June 2nd, six days after the onset of the attack, she suffered greatly from vertigo, nausea and vomiting. This attack gradually subsided during the following two days, and on June 5th, she arose from her bed occasionally, but was unable to walk without staggering, requiring assistance in moving about the room. This condition of disturbed equilibrium persisted for about seven days without other symptoms referable to the ears. On June 15th, when attempting to use the telephone, she noticed for the first time what she described as a "peculiar feeling in the left ear," and then discovered she was unable to hear conversation with that left ear. One week later she presented herself for examination, and the hearing tests showed a complete unilateral deafness of the left ear, all tests being referred to the normal right ear. Caloric tests showed a nonfunctioning left labyrinth. Although it was less than a month from the onset of her labyrinthine symptoms no spontaneous nystagmus was present, and compensation was fairly well established. Treatments of some two months' duration with iodids and pilocarpin were unavailing. When seen in October, 1916, the nonfunctioning labyrinth, both static and acoustic, persisted on the left.

Case 2.—A little girl, aged 9 years, developed mumps on February 21, 1918. After an illness of four days, she suddenly had a "terrible noise" in her head, which lasted some minutes and stopped as suddenly as it began. The mother soon noticed that the child did not answer when spoken to concerning her condition, and the child then complained of being unable to hear anything. Examination that same night, February 25th, showed a complete bilateral deafness present but no clinical symptoms of labyrinthine irritation or destruction. At the same time the child complained of her eyes and of headache, stating that "everything seemed blurred and funny looking." No apparent involvement of the eye muscles or pupillary changes were noticeable, and on examination of the fundi the following morning showed only a slight inflammation of the nerve head and some congestion of the disc on the left side, with no abnormality in the right eye. At no time during this attack did the child complain of any vertigo or nausea. Some three months later, May 15, 1918, this patient was seen again and tested in the turning chair and by the caloric method, and normal reactions were obtained from all semicircular canals, showing no involvement of the vestibular branch but a complete paralysis of both cochlear branches still present. A second eye examination at this time was reported as normal. Deafness persisted despite intensive treatment.

Case 3.—A young boy, aged 5 years, of Italian parentage, had been taken to the Pediatric Clinic at Stanford University Clinic in July, 1919, and a diagnosis of acute epidemic parotitis made there, the disease affecting the left gland only at that time. The usual home treatment was carried out by the mother, a very intelligent type, and the attack subsided after ten days. On the sixteenth day after the beginning of the trouble the boy complained of "some trouble like noise" in his head, not referable to either ear but causing him a great deal of annoyance. When put to bed that night he still complained of the head noise but soon fell asleep. The following morning, when awakened for some medicine, he put his hand to his right ear and told his mother he could not hear. The mother washed out his ears, removed a small amount of cerumen, and dismissed the matter for the time. She noticed repeatedly

during the following week that the child did not seem happy and kept putting his hand to his right ear. The case was then referred to me by a colleague, and examination on July 24, 1919, showed a complete unilateral deafness of the right ear, with slightly impaired hearing in the left. No symptoms of labyrinthine involvement were present nor had the child previously complained of vertigo. Another symptom of much interest and importance, was a hyperosmia, a hypersensibility of the olfactory sense. Young as he was, the child could detect the faintest odors and certain of these were objectionable to him. This condition persisted some weeks, gradually wearing off until normal again. No labyrinthine tests were obtained in this case, the patient leaving for parts unknown without notice. No eye symptoms present.

Case 4.—A young school girl, aged 11 years, very bright and standing high in her class work, was stricken with acute epidemic parotitis with involvement of both glands, in November, 1919. On the third day of her illness, she complained of buzzing in both ears and also of some pain as well. This pain was of the neuralgic type, being intermittent and of very short duration, radiating from the ears to the back of her head in the occipital region. Heat was applied by her attending physician with some relief. The buzzing continued, however, and when seen on November 20, 1919, the ear examination was then negative and the hearing normal in both ears. Despite due precautions, two days later, the patient stated she could not hear very well while lying with her right ear in the pillow. A second examination on November 24 showed a very different result from that made four days previously. A complete unilateral deafness of the left ear existed, the right ear being normal. During the four-day interval between examinations, two other important symptoms occurred, headache of basilar type and blurred vision. Vestibular tests showed normal functioning labyrinths on both sides.

Case 5.—A female adult, married, aged 35 years, was taken ill on October 15, 1921, and a diagnosis of mumps made by her attending physician. The disease began in the right gland and twelve hours later involved the left one as well. The right parotid was more swollen and painful than the left. On October 22, one week after the onset of the attack, the

patient noticed a ringing in the right ear, and some ten hours later some difficulty in hearing when using the phone, which stood on the right side of the bed, necessitating the use of the right ear. On the following day, the patient was suddenly seized with severe vertigo and nausea and she was unable to sit up in bed or stand on her feet without falling to the right side. This condition was greatly relieved by assuming the horizontal position and closing the eyes, although she had no turning sensation at any time. When seen on October 24th, ten days following the onset of her attack, the right gland was still somewhat swollen and painful, the right ear was completely deaf, vertigo and nausea present when standing or attempting to walk, no spontaneous nystagmus present, no spontaneous pastpointing, and the tinnitus practically gone. The patient complained of severe headache and of another symptom (previously mentioned in Case 3), hyperosmia, her sense of smell being extremely keen. The vertigo gradually diminished and the patient was up on her feet and about again on November 2, 1921. This patient refused to permit the vestibular tests so same were not obtained. On January 5, 1922, and April 14, 1922, when re-examined, the right ear was still completely deaf and the patient still experienced some vertigo when dancing and when leaning over to put on her slippers. No eye symptoms were present except a marked myopia, which was present prior to her attack in October, 1921.

Condensing the important points of these five cases, we find: (1) three children and two adults, (2) four female and one male, (3) four unilateral and one bilateral deafness, (4) three without labyrinthine symptoms (vertigo, nausea, etc.) and two with such symptoms; (5) one with a permanent non-functioning labyrinth, two with normal labyrinthine reactions and two not tested, (6) four with tinnitus or head noises and one with "peculiar feeling" in the ears, (7) three with definite headache and one with pain radiating to the base of the cranium, (8) two with hyperosmia and one without (two cases having been seen before the symptom was noticed) and (9) two cases had definite temporary eye symptoms and two without same (Case 1 not examined for same).

In reviewing the literature on this subject, I have failed to find any mention made of the last three symptoms given above.

namely, headache, hyperosmia and those referable to the optic nerve. It may, therefore, be of some advantage to consider these in connection with the auditory lesion, when attempting to determine what anatomic condition is responsible for the deafness found.

Among the various complications of mumps which have been reported, that of meningitis has been mentioned as occurring but has been considered as one of the less frequent. The question that comes to our mind is, "What type of meningitis do we have as a complication to mumps? What is the organism causing it?" If we can have a meningitis due to the pneumococcus, meningococcus, streptococcus, etc., why is it not possible to have a meningitis due to the infectious organism of mumps, whatever that causative organism may be? A mild meningitis of serous variety or type would have the symptoms—abrupt onset, headache, nausea and vomiting, vertigo, hyperesthesia, ataxia, pareses, strabismus, disturbances of vision, disturbances of hearing, etc., or any combination of these. If the meningitis be of the more severe type we would look for convulsions, coma, opisthotonus, etc.

If we take the symptoms found in the cases reported in this paper and consider them in the light of possible meningeal symptoms, we can explain far more easily the involvement of the various cranial nerves than to accept the theory that the lesion is purely metastatic and affects each individual nerve separately. In our studies of other cranial conditions, such as luetic infection, we know that the auditory is the most vulnerable of the cranial nerves and would therefore be the first involved in any condition affecting these nerves. In a serous meningitis, the group of nerves lying so close in the region of the base would undoubtedly be the first ones affected by such a process, and the more resistant ones would ultimately recover while the more susceptible auditory nerve would be permanently destroyed, in part or in whole.

It is with regret that this possibility of a meningeal origin did not occur to me while the reported cases were under my observation, as much more valuable data might have been obtained at that time, but the desire to bring this matter before those interested in this work with the urgent plea for further investigation in an attempt to prove this meningeal origin,

causes me to offer the following suggestions: (1) An early spinal puncture and laboratory examination in cases of mumps with ear symptoms, headache, etc.; (2) a careful fundus examination and record of the visual field; (3) careful examination for an involvement of the other cranial nerves, especially the motor oculi, olfactory, facial and abducens. The condition is not as uncommon as thought, and much valuable information can be obtained if those seeing these cases will act promptly along the suggested lines.

516 SUTTER STREET.

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LXXVII.

CONGENITAL ATRESIA OF THE POSTNASAL ORIFICES.

BY JAMES E. LEBENSOHN, M. S., M. D.,

CHICAGO.

Congenital occlusion of the choanæ is rare, but of extreme interest because correct diagnosis may often be a matter of fundamental importance. Richardson¹ considers this as one of the most frequent types of congenital malformation of the nasal chambers. He states: "Many of this class of cases are unrecognized at birth, and when speedily dying as a result of the obstruction to respiration are placed under the general class of asphyxia neonatorum. The number of observed cases has increased greatly in the past twenty years, a tribute to the increased number of intelligent workers in rhinology; and to the knowledge more widely diffused that such a deformity may occur."

Among children, many of these cases come to the physician with a history of nasal obstruction, and are diagnosticated "adenoids." Kirby² reports two cases in both of which tonsils and adenoids had been removed and later secondary adenoidectomy had been done.

RETROSPECT.

The first recorded case appeared in 1830 and concerned a fetus (Otto). In 1854, an operation was reported on the first case noted in the living (Emmert). The total number of cases in the literature in 1886 was 17 (Hubbell); in 1898, 21 (Clark); in 1904, 61 (Schmiegelow); in 1910, 115 (Fraser). Since 1910, I find in the archives record of 54 cases. The present total, inclusive of the case I now report, is thus 170. Of special interest to the obstetrician and the pediatrician are those cases that have been observed shortly after birth.^{3 4 5 6 7}

PATHOLOGY.

Congenital choanal atresia has been reported three times as often on both sides as on one side alone, and the obstruction

when present is usually complete. In 90 per cent of the cases the occlusion has been of the osseous type. The obstructing plate of bone is vertically placed, has a concave pharyngeal aspect, and is situated within the nasal chamber, the posterior edge of the vomer extending about a millimeter beyond it.

The central portion of the occlusion is thinner than the periphery and may sometimes be composed of membrane only. In this case postnasal examination generally shows a more or less marked dimpling at this point (Fig. 1)—occasionally a perforation. However, this opening, when present, is functionally of little value, as an aperture the size of a crow's quill gives the patient neither the ability to blow the nose nor to respire.

The accepted embryologic explanation of the typical form of congenital choanal atresia just described is this: The primitive nasal cavities commence as blind cul-de-sacs. These cavities elongate, and finally the obstructing nasobuccal membrane gives way. In case of choanal atresia, however, this membrane has become permeated by mesoderm and so fails to break down.

CHOANAL OCCLUSION IN THE NEWBORN.

The symptom complex of choanal occlusion in the newborn is quite different from that in the adult. In the newborn, bilateral occlusion of the choanae constitutes a definite emergency, and the life of the infant may depend upon making the diagnosis. The distinctive symptom is "cyclic dyspnea" (Richardson).⁴ The child struggles for air, the face becomes slightly cyanosed, the child cries, and the mouth breathing instituted relieves the distress. The child rests, and the cycle of respiratory difficulty recurs. So great is the dyspnea as to prevent the infant from taking either the breast or the bottle. Since mouth breathing is an acquired habit, the newborn child thus affected suffocates unless special measures are taken.

Thick, gelatinous, clear mucus fills the nasal cavities—another characteristic, pathognomonic of this affection.

To verify the diagnosis:

1. Syringe the cavities. The fluid does not enter the pharynx but returns in excess at the anterior nares.

2. Pass a probe. The resistance of the obstructing osseous plate is felt.

In the management of this emergency, the immediate object is to secure and maintain mouth breathing. By pressing the lower lip down the dyspnea is relieved. The lips are thus kept apart, day and night, by a relief of nurses, until the end of the second week, by which time the child has probably learned to breathe through its mouth. During this period nutrition is maintained by dropper feeding. The child shortly learns to breathe through its mouth and suck at the bottle alternately.

After the ninth month the nasopharynx is sufficiently well developed to permit of the finger manipulation necessary to operation. To prevent possible alterations in the facial skeleton the operation should be undertaken at this period or soon thereafter.

CHOANAL OCCLUSION IN LATER YEARS.

Choanal atresia in later years likewise produces a very definite clinical picture. On the side of the choanal closure the following constant phenomena are observed:

1. Nasal obstruction of the completest type. Neither respiration nor expulsion of mucus is possible. In crying, the tears flow out of the anterior nares, owing to their inability to pass into the pharynx.

2. Anosmia occurs because of the absence of an air current. After removal of the obstruction the sense of smell is sometimes partially regained.

3. Accumulation of a distinctive type of mucus, unknown in any other nasal condition. The mucus is thick, albuminous, extraordinarily tenacious, and of a peculiar bluish color. Because of retention it is sometimes offensive, and by irritation may occasion a regional dermatitis. The patient, by pressure against the alæ, may be able to remove the viscid secretion.

4. Atrophy of the turbinals, makes the nasal cavity appear more spacious than usual. The posterior tip of the inferior turbinate may be wanting. The mucosa is very pale, sodden and inelastic; and the inferior turbinate may show mulberry degeneration (Fig. 2). The atrophy is due to lack of use and to the pressure of the masses of mucus.

5. Deviation of the septum toward the obstructed side in unilateral cases. Several causes combine to produce this effect:

(a) The occlusion holds the septum to its side (well shown in X-ray, Fig. 3).

(b) The obstructed side is not subject to the dilating influence of the air current.

(c) The V-shaped groove in the upper border of the vomer may be maldeveloped on this side, so that the cartilage has slipped out and projects under the mucous membrane.

6. History of difficulty in maintaining nutrition in infancy. Among variable symptoms that may or may not be present are: Other anomalies of developmental arrest; mental deficiency; nasal voice; moderately separated lips;—in bilateral cases, auditory involvement and disturbance of taste;—in unilateral cases, facial asymmetry, unilateral sweating and inequality of the palatal arch (the arch being higher and narrower on the side of the choanal occlusion).

THE OPERATION.

The usual technic has been to operate under general anesthesia. Experience with the local anesthetics in this operation has not been satisfactory. With the finger in the pharynx as a guide, the occluding bone is removed by the electric burr or with chisel, punch or curette. Special care is taken to remove entirely the projecting border of the vomer and to smooth down nicely the edges of this newly made aperture, for otherwise recurrence of the closure by granulations will take place. The operation is easily done and without much hemorrhage.

In the after treatment, Thomson⁸ relies on simple syringing, but I have not found this sufficient to keep down the granulations. I would recommend the method of Brady.⁹ He finishes the operation by passing around the septum a strip of bismuth impregnated gauze, and tying this around the columella. This is changed daily by attaching a fresh strip of bismuth impregnated gauze to one end of the old strip and then pulling on the free end. The change is painless and is kept up for ten days.

Another method for preventing the growth of occluding granulations from the vomerine border is to combine the re-

moval of the obstruction with a thoroughgoing submucous resection of the septum.¹⁰ This procedure is all the more justified, as a marked deviation of the septum is always present in this condition.

The use of rubber tubes in the after treatment occasions an irritating discharge and is not recommended.

CASE REPORT.

The patient—an electrical worker, white male, 23 years old, of good muscular development, weighing 144 pounds, and 5 feet 7½ inches tall—was admitted to the U. S. Veterans' Hospital, Maywood, Ill., July 19, 1922, for weakness in the right leg. The diagnosis was "thrombophlebitis of the right femoral vein following pneumonia contracted while in the U. S. Naval Service."

This man came under our observation in the routine otolaryngologic examination which every patient admitted to the hospital receives. He was phlegmatic in temperament, had a grammar school education, and was of average mentality. The patient volunteered no complaint of nasal trouble, but on questioning admitted difficulty of breathing on the left side. He had twice been "operated on for adenoids" without relief and had become resigned to his nasal condition.

Examination: The right side of the face is slightly fuller than the left (Fig. 5).

Anterior rhinoscopy reveals a normal right naris with the turbinals well developed and moderately turgescient.

The left naris is filled to a considerable extent with a clear tenacious mucus. Later examination showed that this mucus was most abundant in the morning and that by evening the patient had wiped away most of the secretion. The appearance of the turbinals in this naris was striking. The inferior turbinal showed an advanced degree of mulberry degeneration (Fig. 2). The mucosa was anemic and sodden. Owing to the undeveloped condition of the turbinate bodies, this nasal chamber appeared much roomier than the right (though it really was much smaller—note skiagram, Fig. 4). The septum was free from spurs and appeared to anterior rhinoscopy only slightly deviated to the left.

Postnasal examination was facilitated by a roomy and almost insensitive pharynx. Even a laryngeal mirror could be used and the case easily demonstrated to the most inexperienced nasal examiner. The right choana was without pathology. The left choana was distinctly smaller than the right, and about one millimeter external to the vomerine border was the occlusion shown in Fig. 5. In the center was an invagination that measured 1 mm. transversely, and $2\frac{1}{2}$ mm. vertically. It appeared like a perforation, but syringing the nostril with methylen blue tinted fluid revealed no passage. A transilluminated view of the occlusion was secured with a Cameron diagnostic lamp in the left naris. The osteomembranous character suggested was subsequently confirmed by operation.

Testing the inspiratory and expiratory blasts demonstrated absolute blockage on the left side. Likewise anosmia on this side was complete.

The only other abnormality with the nose and throat was a bifid uvula, the left division of which was the longer. The palate was normally arched with the raphe in the midline.

Both ears were alike without pathologic process and responded with equal normality to all tests. In these unilateral occlusions it has been repeatedly observed that hearing is not affected. This finding discredits the idea that the common forms of intranasal obstruction, such as spurs, deviated septum, etc., are a determining factor in impaired hearing. However, they may be an indirect influence by localizing catarrhal processes to the eustachian tube of the affected side.

The skiagrams (Figs. 3 and 4) demonstrated clearly on the affected side the undeveloped nostril, the atrophied turbinates, the bony periphery of the occlusion and the marked posterior deviation of the septum. The accessory nasal sinuses were all clear.

Communication with the parents in regard to the early history of the patient elicited the following interesting statement: "As an infant he had a discharge from his nostrils and nursed with difficulty. When he was about two weeks old something green and about an inch long came out of his right nostril on sneezing. We thought the trouble with his nose was a cold and applied hot towels as our doctor instructed, but the left

nostril never became free." A photograph of the patient as an infant shows the face to be perfectly symmetrical (Fig. 6.).

The operation was performed under light ether anesthesia and completed in five minutes. Simply, the bony occlusion was removed with the posterior border of the vomer and the edges of the newly made aperture smoothed. No packing was introduced. The after treatment consisted only of cleansing sprays. The recovery was rapid. The patient could now breathe as freely through the left nostril as through the other. The sense of smell, too, was partially regained. Strong odors, such as turpentine, were now recognized, though not tobacco or more delicate scents.

A letter from the patient six months later informed me that a sense of obstruction was developing in the operated nostril and that the mucus, which was once more continually being formed, was becoming increasingly difficult to blow out.

I am hoping that I may have the opportunity to operate once more on this case. In this event, after excising the granulations that have formed, I shall remove the posterior part of the septum by submucous resection. This, I believe, should prevent a recurrence of the obstruction.

3159 W. ROOSEVELT ROAD.

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Fig. 1. Congenital occlusion of the left posterior choana. The black area in the center represents the membranous portion that appears dark because invaginated.



Fig. 2. Mulberry degeneration of the left inferior turbinate. The degeneration is probably due to the constant irritation of the accumulating mucus.

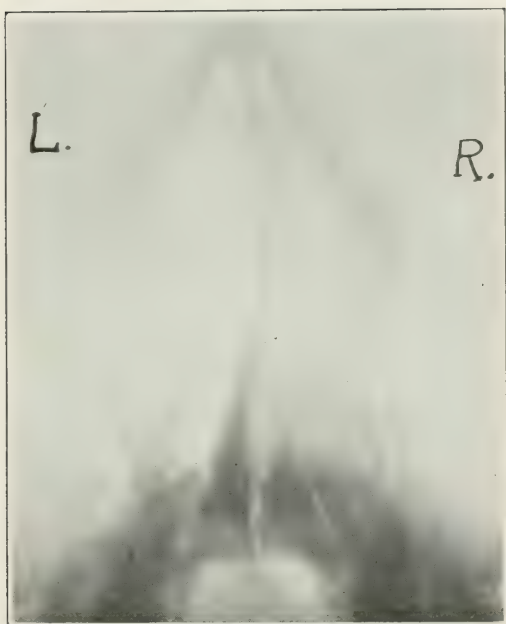


Fig. 3. Skiagram showing posterior deviation of the septum, and atrophied condition of turbinates in left naris. The small size of the left posterior choana is very well indicated.

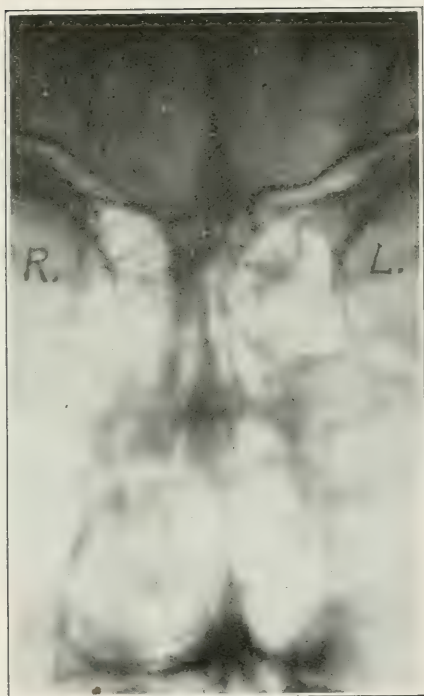


Fig. 4. Skiagram showing the undeveloped left naris with its undeveloped turbinates. The osteomembranous condition of the occlusion is better suggested in the original film.



Fig. 5. The patient at age 19. The right side of the face is somewhat fuller than the left.



Fig. 6. The patient as an infant. The face appears entirely symmetrical.

LXXVIII.

NONTRAUMATIC HERNIA OF THE DIAPHRAGM. AN EMBRYOLOGIC VIEWPOINT.*

BY LYMAN G. RICHARDS, M. D.,

BOSTON.

With the recent advances in roentgenologic diagnosis and the constant perfection of operative technic the problem of diaphragmatic hernia has become more and more a surgical one, resulting, in the nontraumatic cases, in the persistent neglect of their etiology, their commonest points of occurrence and their relation to the normal landmarks of the diaphragm. The confusion and uncertainty underlying this surgical enthusiasm is well shown by a quotation from one of the leading surgical journals. "Congenital hernias do not need to be present at birth; hernias should be considered congenital, when they are formed, because there is a weak point in the diaphragm. . . . This is generally situated around the esophagus. . . . We do not know if there are cases of hernia through the opening for the aorta or vena cava. . . . Diaphragmatic hernias differ from all others because . . . they are deprived of an enveloping sack." Another author states that "false hernias are always traumatic." These statements indicate not only uncertain but inaccurate knowledge of the fundamental principles of diaphragmatic hernia. Surgeons generally assume certain weak spots to be the sites of hernia, regardless of where these are or why they occur.

I have undertaken in this article to correlate the embryology of the diaphragm, so far as this is definitely understood, with the subject of nontraumatic hernia, explaining the frequency of the various types and their points of occurrence, in the light of what is known of the elements which compose the diaphragm. I have also collected all the nontraumatic

*The investigation of this subject was made at the suggestion of Dr. Harris P. Mosher, Professor of Laryngology, Harvard Medical School.

cases reported since 1900 and tabulated those reported previously by Thoma and Grossa (Fig. 11 and tabulation).

ANATOMY.

The adult diaphragm is a musculofibrous septum arising from the circumference of the thorax. It is elliptical in shape, composed of a peripheral muscular portion and a central tendinous portion. It is attached in front by fibers to the ensiform cartilage, on either side to the inner surface of the cartilages and bony portions of the six or seven inferior ribs, interdigitating with the transversalis muscle, and behind to two aponeurotic arches, the internal and external arcuate ligaments. Centrally it is attached to the lumbar vertebræ on each side by its crura. There is a muscular deficiency on either side of the slip from the ensiform cartilage, filled by areolar tissue covered on the thoracic side by pleura and on the abdominal side by peritoneum. These deficiencies form the so-called foramina Morgagni or Larry's spaces. There are also similar triangular areas devoid of muscle posteriorly between the fibers arising from the internal, and those arising from the external, arcuate ligaments known as the foramina Bochdalecki.

The central tendon of the diaphragm is a thin strong aponeurosis shaped like a trifoil situated immediately below the pericardium, with which it is partly blended.

The openings connected with the diaphragm are three large and several smaller apertures. The former comprise those for the aorta, the esophagus and the vena cava. The aortic opening is the lowest and most posterior of the three large apertures, being on a level with the first lumbar vertebra. It is slightly to the left of the midline, immediately in front of the bodies of the vertebra and hence, strictly speaking, it is not in the diaphragm at all but behind it, the anterior margin being formed by a tendinous arch thrown across from the crus on one side to that on the other. It transmits the aorta, the vena azygos major and the thoracic duct. The azygos major may pass through the right crus.

The esophageal opening is situated at the level of the tenth dorsal vertebra. It is elliptical in form, muscular in structure and formed by the decussation of fibres from the two crura. It is anterior to, and a little to the left of, the aortic opening

and transmits the esophagus, the two vagi and small esophageal vessels.

The opening for the vena cava is on a level with the disc between the eighth and ninth dorsal vertebræ. It is quadrilateral in shape, lies entirely within the central tendon and transmits the inferior vena cava.

The right crus transmits the greater and lesser splanchnic nerves of the right side, the left crus those of the left side together with the vena azygos minor.

The diaphragm is covered above with the right and left pleura and the pericardium, below by the peritoneum. It is in relation on the right with the right lung and the liver, on the left with the left lung, stomach, spleen and left kidney.

The phrenic nerve, originating from the fourth and fifth branches of the cervical plexus, in conjunction with the fifth and sixth intercostals, supplies the diaphragm. The left phrenic nerve is longer than the right, since the left half of the diaphragm is lower than the right and because the heart protrudes on the left side.

COMPARATIVE ANATOMY.

The only animals showing a complete formation of a true diaphragm are the mammals. In the lower forms it is only the pericardial cavity which is separated from the abdominal, and there may even be a persistent pericardioperitoneal canal connecting them. In some urodeles there is found an analogous but not homologous muscular structure springing from the vertebral column and ribs which forms a partition between the pleural and peritoneal cavities but which is complete on both sides only in crocodiles. In this partition there is no evidence of a central tendon; and Keith alone upholds any homologous relation between the two structures.

EMBRYOLOGY.

In the early human embryo the pericardial, pleural and peritoneal cavities communicate, all three leading indirectly into the extra-embryonic coelom. At this time the pericardial cavity is a paired structure except anteriorly, where each side is connected with the other to form a U shaped cavity. In this cavity the heart develops and with further growth

the splanchnic mesodermal layers are folded together to form the ventral wall of the pericardial cavity (Figs. 1 and 2), being attached for a short time to the heart by the ventral mesocardium, a structure which soon disappears to leave the heart free at this point. Passing forward toward the heart on the splanchnic layer of the mesoderm covering the yolk stalk are the large vitelline veins, one on either side, converging to an acute angle to form the sinus venosus. This angle between the veins is filled with a solid mesodermal structure forming the ventral mesentery and first called by His the septum transversum. This structure forms the earliest anlage of the future diaphragm (Fig. 3). The vitelline veins soon become so large and expand so far laterally that they bring the splanchnic mesoderm in which they lie in contact with the somatic mesoderm which forms the lateral wall of the pericardial coelom (Fig. 4). Fusion of the two layers of mesoderm along the course of the veins now takes place, and each lateral coelomic or parietal recess becomes divided into two parallel passages, a dorsal and a ventral parietal recess (Fig. 4). The angle at which the veins unite and their lateral divergence result in the closure caudally of the ventral recesses and in the interruption of their continuity with the trunk cavity, so that they form two blind pouches extending a short distance below the mesodermal mass in which the juncture of the veins lies and which forms the floor of the pericardial cavity. The dorsal recesses, on the other hand, retain their connection with the trunk cavity by way of the so-called pleuropericardial canals for a much longer period.

This fusion of splanchnic and somatic mesoderm laterally, together with the transverse septum, forms a thick semilunar mass which projects horizontally into the coelom with its free edge facing dorsally. Laterally it is continuous with the body wall, mesially it is continuous with the dorsal mesentery, a structure which connects the pericardium with the vertebral column and which contains the foregut. The latter is connected by the ventral mesentery to the anterior body wall, and into this mesentery, caudal to the septum transversum, grows the liver. This structure pushes into the substance of the septum, which thus acquires a considerable thickness, especially toward its dorsal edge (Fig. 5A). It furthermore be-

comes differentiated into two layers, an upper and thinner, which form the floor of the ventral portion of the pericardial cavity, and a lower and thicker, which contains the liver. In later stages the layer containing the liver becomes separated from the upper by grooves or infoldings of peritoneum, which gradually deepen medially and dorsally, but without actually reaching the midline, a portion of the septum remaining as a midline fold or the adult falciform ligament, the free edge of which contains the umbilical vein (Fig. 5B). The failure of these grooves to separate the liver completely dorsally gives rise to the permanent coronary or falciform ligament.

Thus there is formed a ventral separation of the pericardial and peritoneal cavities. A dorsal communication still exists between them through two narrow, short canals, which later give rise to the pleural canals or cavities. The further separation of the pericardial cavity from the two pleural cavities, and of these in turn from the peritoneal cavity, is an extremely complicated process and one concerning which embryologists differ, but it is the closure of this portion of the communication with which we are most concerned in hernia of the diaphragm. The study of this procedure is rendered all the more difficult by the fact that there is a constant increase in the flexion of the embryo, such that the pericardial cavity comes to assume a ventral instead of a cephalic relation, thus making serial sections confusing. The chief factors in the further closure of the dorsal canals are the Cuvierian ducts. They are the result of the union of the anterior and posterior cardinal veins developing in the lateral body wall to form the common cardinals which gain entrance to the heart through the fusion of the splanchnic and somatic mesoderm described above. The protrusion of the ducts toward the dorsal coelomic cavities causes, first of all, a diminution in the size of the latter and then gives rise to a circular ridge, called by Mall the pulmonary ridge, extending from below on the dorsal wall near the top of the Wolfian body, upward, laterally, anteriorly, and then caudally to the free edge of the transverse septum. This ridge later broadens and its edges become thicker and more prominent than its central portion in such a way that a triangular protrusion is formed, the cranial and caudal margins forming the sides,

the dorsal body mesentery the base of the triangle (Fig. 6). The cranial margin or side of this protrusion forms the pleuropericardial membrane, and its further extension and growth on each side reduce the communication between the pericardial and pleural cavities to a mere slit. The caudal margin becomes the pleuroperitoneal membrane and eventually separates these cavities.

In two-millimeter embryos the septum transversum lies about opposite the middle cervical region, in a transverse position. According to Mall, there is a gradual migration caudally until at 24 mm. it is opposite the first lumbar segment. It is at the time when it is opposite the fifth cervical segment that the septum receives the phrenic nerve. With this descent there is a reversal of position almost at right angles to its earlier one, so that the original dorsal surface becomes ventral. This descent also serves to carry the pleuroperitoneal membrane caudally, in such a way that the lung, which is meanwhile developing from the walls of the dorsal mesentery, comes to lie in the triangular space between the pleuropericardial and pleuroperitoneal membranes (Fig. 7). The latter is still unclosed, and for a time the lung projects below it into the abdominal cavity. With the descent of the septum the dorsal end of the pleuropericardial membrane lags behind and thus comes to assume a position nearly at right angles to the septum. Since the communication between the pericardial cavity and the pleuroperitoneal canals is greatly narrowed by a general expansion of the peripheral tissue—i. e., laterally by the pulmonary ridges, medially by a thickening of the dorsal mesentery to form what Uskow calls the longitudinal mass, it is a simple matter for the pleuropericardial membranes to fuse with this longitudinal mass and thus cut off the pericardium completely. It is not till somewhat later that the pleuroperitoneal membrane fuses with the dorsal mesentery, thus separating the peritoneal cavity from the pleural cavities. The lungs have by this time receded and the septum descended sufficiently to include them within the pleural cavities.

The lungs begin to develop in the longitudinal mass on the medial walls of the pleuroperitoneal canals, into which they bulge laterally. The right lung develops further down in the

pleuroperitoneal canal than the left. At the time of closure of the pleuroperitoneal openings the membrane accomplishing this is so placed that its lateral half extends much further cephalic than its medial half, so that a transverse section through it would show peritoneal cavity external to and above the pleural cavity, a relation exactly opposite to that in the adult. This early relation is reversed by the caudal growth of the lungs, which push the diaphragm laterally until a section shows the pleural cavity laterally and the peritoneal medially.

Growth of the lungs laterally is responsible for the further development of the true pericardium, which is at first closely bounded on each side by mesoderm of the body wall of which it is really a part (Fig. 8A). Later the lungs push forward around the pericardium, splitting off a thin layer which forms the real pericardial sack covered laterally with parietal pleura (Fig. 8B). The phrenic nerve first grows between the subclavian veins and cardinal veins along the lateral wall of the pleural coelom. With the subsequent separation of the pericardial from the pleural coelom by the growth of the pleuropericardial membrane, the nerve is left on the lateral edge of this separating membrane. Later when the lung burrows to extend around the side and front of the heart the nerve is pushed into the pericardial wall between the heart and the lung.

The above description is concerned only with the membranous separation of the three body cavities. The adult diaphragm is largely a muscular structure, the full development of which takes place much later. The muscle elements invade the diaphragm while it is still high in the neck, originating mostly, according to Sobotta, from the fourth cervical myomere. The muscle grows in from the lateral body walls to a certain point, after which its further centripetal extension is accomplished by pressure from the expanding lungs, which peel off the muscle along the body wall as they advance downward. In early fetal life the entire diaphragm, like the septum of some animals, is composed of muscle, and the tendinous portion originates only secondarily by an atrophy of the central part of the muscle.

Another structure of embryologic significance in the study of diaphragmatic hernia is the superior omental recess. This is a cranial projection of the general abdominal cavity, at first bilateral, on either side of the dorsal mesentery of the gut. This fingerlike extension on the left side is obliterated at an early stage in development, but that on the right side persists for some time and eventually gives rise to the superior omental bursa (Fig. 9). At one stage in its development its extreme upper or cephalic portion becomes narrowed and cut off at the level of the esophagogastric union to form a blind pouch. Normally this either disappears by obliteration or persists as a small bursal sack enclosing the esophagus on all sides except at its mesenteric attachment posteriorly, just as the testicle is enclosed in the tunica vaginalis. In rabbits this bursa, called the infracardiac bursa, is constant; in adults it is usually not evident. It may be entered by splitting the leaves of the ligamentum pulmonale, just below which it lies, whereby a sound can be passed directly through the diaphragm into the abdominal cavity and lesser sack as in a congenital inguinal hernia.

Thus the adult diaphragm is derived from a number of separate elements which, in the order of their appearance, are as follows (Fig. 10):

1. Septum transversum (ventral mesentery). Gives rise to the ventral and central portion of the diaphragm, especially the part directly below the heart.
2. Dorsal mesentery. Gives rise to the posterior and central part containing the esophagus.
3. Pleuroperitoneal membrane. Gives rise to the postero-lateral portion of the diaphragm where the costal and lumbar segments meet.
4. Septum transversum (lateral extensions). Gives rise to costal portion on each side.
5. Thoracic wall (peripheral part, especially laterally).

It is a difficult matter to obtain a clear mental picture of the processes involved in the union of these varied elements, the more so since they take place in all three planes at a time when the embryo itself is constantly changing its shape and relations. A helpful if not entirely accurate simile is that suggested by Waldeyer,¹ in which he describes the separation of

the coelomic cavities in terms of the nasopharynx, an anatomic field which is much more readily visualized than the developing body cavities. He conceives of the anterior nares as closed and the anterior portion of the nasal septum absent. This single cavity then represents the pericardium. Prolong the septum backward till it crosses the nasopharynx and touches the posterior pharyngeal wall, thus dividing the pharynx into two parts representing the two pleural cavities which become united below the lower edge of the septum with the esophagus, the latter representing the peritoneal cavity. Approximate the tongue to the hard and soft palate, the tongue representing the liver, the hard palate the septum transversum and lateral mesocardium, or together, the ventral component of the definitive diaphragm. The soft palate is the pleuro-pericardial membrane which, passing from the posterior part of the ventral diaphragm, finally fuses with the posterior pharyngeal wall to separate the nasal cavity from the two pharyngeal or pleural cavities. The tonsillar pillars or pulmonary ridges are to be conceived of as originating below on the posterior pharyngeal wall and as passing upward and forward in a concave arch to fuse with the "dorsal diaphragm" or soft palate, forming a crescentic lateral ring or groove from above downward and backward. Thus a triangular shaped area is outlined by the soft palate and pillars, the gradual narrowing and constriction of which, together with the lateral thickening of the septal mass, closes off each half of the pharynx from the esophagus or each pleural cavity from that of the abdomen.

The foregoing account of the embryologic development of the diaphragm is in accordance with the views accepted by the majority of embryologists. Keith,² however, has a quite different concept, based on, and supported by, analogies from the point of view of comparative anatomy. According to him, the pleural cavities were never a part of the general coelomic cavity but were developed in the body wall by extrusion of the lungs, much as the testicles are extruded from the abdominal cavity, retroperitoneally, the internal abdominal ring corresponding to the pleuroperitoneal opening. He found that in amphibians the lungs were in the abdomen below a diaphragm composed of the transverse septum (analogue of the mamma-

lian ventral diaphragm), and a fascial layer of cervical aponeurosis. This diaphragm is supplied with muscle derived anteriorly from the rectus, posteriorly from the cervical portion of the transversalis. In reptiles this "abdominal" lung pushes up and evaginates the whole dorsal fascia over it. In birds and mammals it pushes up a part and then perforates it centrally through an opening which, according to Keith, is the pleuroperitoneal opening, which may or may not close, just as the processus vaginalis may or may not close. As soon as the lung is above the dorsal fascia, with its layer of transversalis muscle, it pushes its way forward into the transverse septum in such a way as to separate this into a pleuropericardial part and a pleuroperitoneal part, the latter being pushed down to form the dorsal part of the definitive diaphragm.

This idea is well supported by the facts of comparative anatomy. Actual perforation of a tissue layer is, however, a rather unusual embryologic principle, and it is in this respect that the analogy of the descent of the testicle ceases, since this organ is from first to last retroperitoneal and never perforates a layer. Also this theory does not help to solve any of the problems of diaphragmatic hernia and assumes a contemporary formation of pleuropericardial and pleuroperitoneal membranes, which so far as their closure indicates, is not in accord with the facts in mammals.

CLASSIFICATION OF DIAPHRAGMATIC HERNIAS.

The lack of any uniform method of classification of these hernias has led to a great discrepancy and disagreement as to the real meaning of the terms "congenital" and "acquired" in the statistics reported by different authors. Surgeons have divided them surgically and judged the frequency of the various forms solely from their incidence at the operating table, while anatomists have classified them anatomically on the basis of autopsy findings.

The following classification combines most satisfactorily the essential features of this form of hernia:

1. True hernias (those with hernial sack).
 - A. Congenital (present at birth).
 - B. Acquired.
 - a. Through the natural openings (mostly esophageal).
 - b. Elsewhere (traumatic or nontraumatic).

2. False hernias (those without hernial sack).

A. Congenital.

B. Acquired (all traumatic).

3. Eventration of diaphragm.

This classification does not differentiate between those cases in which a congenital weakness may have been present at birth but in which the hernia did not manifest itself until later in life. Usually it is impossible to make such a differentiation, except in those cases in which no sack is present. If trauma can be excluded, such cases are all congenital. Where any prolonged intraabdominal pressure finally results in a hernia, there is no way of excluding a preexisting hernia.

This article is not concerned with hernias of traumatic origin. They are of interest primarily from the surgical point of view, are all acquired and bear no relation to the development of the diaphragm. They are not, however, all false, as some authors state, as a crush or fall may easily cause a tear in the diaphragmatic muscle without injuring either pleura or peritoneum, giving rise later to a true hernia whose traumatic origin has been forgotten. Such cases may heal and on subsequent discovery be indistinguishable from the congenital form, since the edges of the torn pleura and peritoneum readily grow together and form a smooth edge closely simulating a congenital opening. It is more usual for trauma to rupture both membranes as well as the muscle.

Diaphragmatic hernia has long been known to medical literature, the first congenital case being reported by Riverius in 1689. In 1824 Sir Astley Cooper gave a masterful account of a case. Since then cases have been reported more and more frequently, the condition has been suspected and sought for more often, and the diagnosis more frequently made antemortem. The question of why such hernias occur, why some types are more frequent than others and why some situations are rare and some common, it is the aim of this paper, so far as possible, to answer.

ETIOLOGY OF DIAPHRAGMATIC HERNIAS.

The factors involved in the occurrence of these hernias are as varied as the types themselves and differ according to the location of the hernia. There are, however, certain general

principles governing the occurrence of all forms, which may be divided into two groups, intrafetal and extrafetal.

1. Intrafetal.—These factors are concerned with the development of the fetus itself. The most important is the interference with the normal closure of the pleuroperitoneal membrane, resulting in a smaller or larger defect, usually posteriorly and without a sack. What prevents the closure of this membrane is a much mooted question. That it may be a simple retardation or cessation of growth (as in the case of a persistent foramen ovale) is shown by the frequency with which these hernias are associated with other congenital anomalies, such as a cleft palate, patent ductus arteriosus, other hernias, accessory lungs, and abnormal mesenteric relations of an atavistic nature. In other cases the defect is quite clearly a halt in normal development independent of other factors.

Another frequently mentioned cause is the premature prolapse of abdominal viscera into the thoracic cavity. The reasons given for this prolapse are: (1) abnormal decrease in intrathoracic pressure with a similar increase in intraabdominal pressure; (2) an abnormally long or free mesentery which allows the viscera access to the chest. These reasons do not bear close scrutiny, since it has been shown, first, that so long as the amniotic fluid is present there is no difference between the thoracic and the abdominal pressure, and second, that at the time the diaphragm is being formed the body walls are incapable of sustaining or exerting any pressure whatsoever. Jahn³ has shown that certain normal changes in mutual position and relation of the large intestine and stomach have already taken place before the normal time of closure of the pleuroperitoneal membrane. Since these changes take place only in the abdomen the viscera could not have entered the chest until after the normal time of closure of the membrane, and hence failure of the latter to close was not due to any premature prolapse of the viscera. The fact that still further mesenteric changes normally taking place in the abdomen do not occur in the chest shows that the previous change occurred while the intestine was in the abdomen.

One other factor may increase the intraabdominal pressure and, by exerting pressure on the pleuroperitoneal membrane sufficient to shut off its normally poor blood supply (Vogel),⁴

cause a delay in its development. This is the occasional failure of the small bowel to enter the umbilical coelom, a condition which would considerably increase the abdominal contents at this time.

These factors concern the false type of congenital hernia. The true congenital hernias are due to an abnormal development or formation of the diaphragmatic muscle, and any process which retards or prevents this may easily lead to a herniation at the point of defect. Why the muscle fails at times to develop is not certain. It may be a question of local pathology, some interference with circulation or some fault in innervation. The defect is more usually a pathologic than an embryologic one.

Abnormalities in the growth of the liver have been frequently assigned as causes of a failure in the separation of the two cavities. Since a small liver has been mentioned as often as a large one and since both types have frequently been found accompanying the hernia, it is very doubtful if this organ plays any very important role in the development of the dorsal diaphragm.

2. Extrafetal.—These factors are the same as those associated with many other developmental defects, such as trauma to the mother and intrauterine inflammation. The inverted position of the fetus has been suggested as likely to favor the passage of the viscera from the chest into the abdomen, but such a position is not assumed till long after the normal closing period of the pleuroperitoneal membrane.

SITUATION OF THE HERNIA.

A consideration of any large number of reported cases of diaphragmatic hernia would lead one to believe that there is no portion of the diaphragm which is exempt and no natural opening which cannot, theoretically at least, serve as a point of weakness. As a matter of fact, these hernias fall into several distinct groups, some much more common than others, and there are certain points which never are the site of herniation. Thus the aortic opening has never been known to contain a hernia, first, because this opening is, anatomically and developmentally, not in the diaphragm but behind it;

second, its ring is tendinous, not muscular, and attached closely to the vertebræ by the crura on each side. The aorta is occasionally included in the hernial opening, but this is the result of the extreme defect of the diaphragm on one side and not of any herniation through the aortic opening itself. Similarly the quadrilateral foramen, through which passes the inferior vena cava, has never been found to be the site of a hernia. The intimate attachment of the vein walls to the central tendon and the broad expanse of liver below and around it render access of the abdominal viscera impossible.

The various hernial sites may be divided as follows: Anterior, central, posterior, esophageal, others.

Any of the first three types may be right or left, the latter being much the more frequent, as shown below.

Anterior.—These hernias are situated to one side of the diaphragmatic attachment to the sternum and occur almost entirely through Larry's space or the foramen of Morgagni, one of the so-called points of weakness, where there is a triangular muscular defect between the sternal and costal attachments of the muscle. These are known as parasternal hernias and are usually small defects, being the result of weakness at a point normally somewhat deficient in musculature. The possibility of an incomplete closure of the pericardial ventral recesses when the vitelline veins expand laterally can be excluded by the fact these hernias never communicate with the pericardial cavity. Though the muscle is deficient at birth, these hernias are practically all acquired as the result of a prolonged increase in intraabdominal pressure and are found almost exclusively in adults. The defects may be crossed like a griddle by muscle fibers which occur more commonly and numerously in younger individuals. The ratio of the size of these "spaces of Larry" to the size of the body is, as shown by Thoma,³ practically the same in the newborn as in adults. Why then does this type of hernia not appear in the newborn in which hernias occur at far less favorable sites? There is proportionally less muscle in the adult spaces, the angle of the diaphragm with the sternum is less acute, the mesentery is longer and the liver is relatively smaller, but none of these factors seems to furnish a satisfactory answer. The almost universal presence of a sack renders the problem far different

from that concerned with the closure of the pleuroperitoneal canals.

These parasternal hernias are occasionally bilateral. One has been reported by Lubosch⁶ in which a double sack passed upward on either side of the falciform ligament and extended up into the anterior mediastinum. This is an example of a congenital weakness which became an acquired hernia and is best explained by assuming that when the peritoneum folds in to separate the liver from the diaphragm (Fig. 8B) it meets some abnormal resistance from a thick falciform ligament which deviates the folds upwards toward the thorax to form two peritoneal pockets on each side. Moser reports a case in which the hand could be passed into such a hernia on the left side through an easily reducible epigastric swelling and the entire apex of the heart grasped, undoubtedly through a greatly dilated left foramen Morgagni.

For such of these hernias as have no sack there seems to be only the explanation of a previous forgotten trauma.

CENTRAL HERNIAS.

On first consideration these hernias do not readily lend themselves to any embryologic explanation. They occur both with and without a sack at points unrelated to the fusion lines of the constituent elements of the diaphragm. Those with a sack must be due to some local pathologic condition in the muscle; they are far less common than the false type. These latter are most readily explained by assuming that the posterior part of the pulmonary ridge has begun to develop toward the longitudinal mass and has then for some unknown reason ceased to grow. This would leave a ridge of tissue into which vertebral muscle fibers could advance. As this ridge, together with the rest of the diaphragm, increases in size the defect between the two will move forward and take up a more central position relative to the rest of the diaphragm. If there is no growth at all in the pulmonary ridge then the defect is a pure posterior one occupying the original pleuroperitoneal canal. The direction, the rate and the character of growth of the muscular diaphragm determine the eccentric or central position of these defects and whether they lie in the muscular or tendinous portion. Thus most of the false hernias in the cen-

tral portions of the diaphragm were originally posterior defects which have changed their relative position.

POSTERIOR.

These hernias show more variations in size than any of the others. The majority of them are due to failure in the closure of the pleuroperitoneal canals, hence they originate in the lumbar region between the vertebral and costal portions at the point where the pleuroperitoneal membrane last closes. It has been repeatedly stated that this point is identical with the so-called foramen Bochdalecki in the lumbar triangle, where there is a normal deficiency of muscle much as in the parasternal spaces. This, however, is not the case. This "foramen" is simply a point of incomplete fusion of the vertebral and costal muscle groups and does not appear until long after the pleuroperitoneal canal has closed. That the two do not even occupy the same spot is proved by the occurrence of a posterior membranous defect with a normal "foramen" in a single individual and on the same side, and a case has been reported (Otto⁷) in which a left false and a right true posterior hernia were present simultaneously.

According to Keith,² such posterior defects arise from a failure of the point of perforation made by the lung to close after this organ has made its way upward through the dorsal fascia posterior to the transverse septum.

ESOPHAGEAL.

This situation is regarded by many surgeons as the most common site for diaphragmatic hernia. In the living adult, as seen at operation, it doubtless is, but in the sum total of these hernias the esophageal ones are extremely uncommon. Strictly speaking, there is no "esophageal opening"—that is, the digestive tract is everywhere intimately enclosed by the dorsal, and above the diaphragm, by the ventral, mesentery through which the esophagus passes. The only "opening" is that created by the failure of the diaphragmatic muscle to closely surround the esophagus. The more the muscle fails to do this the greater is the amount of the membranous partition composed of peritoneum, pleura and interstitial tissue which, under certain conditions, may become the site of a hernia. For this reason esophageal hernias are commonly

of the true type, contained in a sack formed of relaxed membranous diaphragm intimately attached to the esophagus itself. Indeed, without the assumption of some forgotten trauma it is difficult to explain an esophageal hernia in which there is no sack present. The great majority of them, as found in adults, are of the acquired type and result from long continued coughing or straining in an individual whose esophageal muscular opening is abnormally large or weak. The congenital cases with a sack may be explained, as suggested by Bund, by an abnormal development of the omental bursa (Fig. 9). If for any reason the infracardial bursa, which at an early stage communicates with the omental bursa and hence with the general abdominal cavity, should persist and remain open below, there would then be formed a direct passage beside the esophagus and through the diaphragm. This would afford an extremely vulnerable spot for a viscus to force its way upward along and close to the esophagus, through the muscular ring, simulating in every way a general dilatation of the opening. Since the stomach would be the organ most likely to herniate by this route, the esophagus would simply be carried upward at the fundus of the sack.

This infracardial bursa persists more frequently on the right than the left, so that we should at first expect that most esophageal hernias would be found in the right chest, but once the opening has become sufficiently dilated to allow the stomach to leave the abdominal cavity other factors control the side to which it goes. Thus the presence of the heart on the left would have a tendency to force the hernial sack to the right, as would also the greater negative pressure in the right thorax. An interesting case in point is that described by Rischbieth⁸ in a newborn child with an encephalocoele and other congenital abnormalities. There was a complete absence of the left half of the diaphragm save for a muscular strand to the left of the esophagus. There was also a huge dilated esophageal opening with a sack containing the stomach and almost all the other viscera which had herniated into the right chest and posterior mediastinum. The question arises why these viscera did not enter the much more accessible left chest which contained only the left kidney. Rischbieth explains the extensive herniation into the esophageal sack by the fact that

in the absence of a left diaphragm the descent of the right diaphragm (which occurs as a result of simple muscular contraction, not as a part of respiration) caused a great increase in the negative pressure of the right side and hence favored the aspiration of the viscera toward that side.

The formation of the sack is somewhat obscure in those cases where its neck is reflected not from the cardia but from the pyloric portion of the stomach, the latter being to all intents a thoracic or mediastinal organ and situated entirely above the diaphragm. In such cases the stomach may never have been in the abdomen, but, developing at an abnormally high point in the digestive tract, may have remained stationary while the diaphragm was formed and closed in its normal position. There is no reason why the pleuroperitoneal membrane might not close posteriorly, even with the stomach in such a position, since its relation to the dorsal mesentery would be entirely normal. Where there is a large sack reflected from the cardiac end of the stomach, the cause is clearly one of herniation from below and is certainly the more common type. The difficulty of reducing many of these hernias suggests a high mesenteric attachment of the stomach when adhesions between it and the thorax can be excluded.

OTHER SITUATIONS.

A few rarer points of exit are by way of the sympathetic nerve chains in the crura of the diaphragm and via the azygos minor vein. Another rare but extremely interesting point of herniation is the floor of the pericardium. Keith⁹ reported two cases of this type in 1910, Martland¹⁰ one in 1908. All three were in adults who had lived for years with the heart surrounded by coils of small bowel without any apparent symptoms. Such hernias, which are of the false type without a sack, result, according to some authors, from a congenital failure in closure of the pleuropericardial membrane. Since, however, there was no communication between the pleural and pericardial cavities, this seems a hardly tenable theory. A more plausible one would explain the defect on the basis of a failure in complete development of the transverse septum, this being the structure which forms the adult separation between the peritoneal and pericardial cavities. Were this earliest en-

largement of the adult diaphragm defective, one would certainly expect a secondary defect in the subsequently formed pleuro-peritoneal and pleuropericardial membranes, structures which were entirely normal in the three cases. As no congenital cases have been reported, more satisfactory explanation is afforded by assuming all these adult cases to be the result of some past trauma which ruptured the central tendon below the heart. This is the explanation assumed by Stoeber in the case of a similar hernia in a dog, because the edges of the opening, instead of being firm, showed a free flaccid margin of pericardioperitoneum which, when brought together, completely closed the opening, an unlikely condition in a defect of congenital origin. Frank defects between the pericardial and pleural cavities have been reported several times, but in none of these was there any communication between the pericardial and peritoneal cavities.

The above classification of the points of herniation does not take into consideration one other frequent condition, namely, a total unilateral lack of the diaphragm. Such cases if examined carefully will almost always show a small sickle shaped fold of muscle anteriorly representing the transverse septum, the defect being due to a complete failure of development of the pleuroperitoneal membrane, which becomes proportionally greater as the growth of the fetus continues. Total lack of the whole diaphragm, which has been reported once, occurred in a monstrosity. That left hernias are far more common than right sided ones is a fact, the explanation for which has long been a subject of controversy, though it is commonly assumed to be the result of the greater protection offered by the liver to the right side of the diaphragm. This undoubtedly plays an important role in the matter, but it has been shown that at the time of the development of the diaphragm the left half of the liver affords the diaphragm about as much protection as the right. The foramen of Bochdalecki is larger on the left than the right, indicating that muscle formation is slower on the left side, but this does not explain the false type of defect. The mobility of the stomach has been considered instrumental in exposing the left side, while the further descent of the right lung into the abdominal cavity may prevent the entrance of viscera. It has been shown above,

however, that a mechanical interference with closure on the part of the abdominal viscera plays but a small part in the etiology of early defects, so that there must be some other factor which at the very outset renders the left diaphragm more liable to incomplete development. Its exact nature must remain one of the unsettled problems of embryology.

CONTENTS OF THE HERNIA.

Lacher states that the only viscera which have not been found at least once in the chest cavity are the urogenital organs and the rectum. The stomach is, of course, the most frequent organ to herniate, the large intestine the next, and the small bowel next. In one instance only has a thoracic organ been found in the abdomen, in which case the tip of the right lung was found between the folds of the falciform ligament. The defect presumably involved the right foramen of Morgagni.

VISCERAL RELATIONS IN THE THORAX.

The extrusion of the abdominal viscera into the thorax has a marked effect on those which normally occupy the chest cavity. The heart is almost always displaced to the side opposite the diaphragmatic defect, and the lung on the same side is pushed upward and hindered in its development, especially where the hernia has existed from birth. It is often a difficult matter to decide whether the appearance of the lung is due to a pressure atrophy or whether there exists a primary failure in lung development of a truly embryologic character. A frequent finding secondary to the presence of the abdominal viscera in the chest cavity is a further herniation through the posterior mediastinal tissues of the organs occupying the apex of the sack. This may be so extreme as to permit viscera which herniated through the left diaphragm to lie entirely in the right chest. The same situation occurs less often in hernias through the parasternal spaces, where a sack originating on the left side may pass across the tissues of the anterior mediastinum and come to lie in the right chest.

It has been frequently observed that in the absence of a hernial sack the prolapsed viscera never becomes adherent to the parietal pleura of the chest walls. Since the transverse

colon, for instance, often lies in the chest at a time previous to that at which, if in the abdomen, it would become anchored at certain points to the parietal peritoneum, there must be some explanation as to why this fusion fails to occur in the thorax. The pleural membrane may be less suited to such a union; constant motion of the heart may be a factor.

PROGNOSIS.

A surprisingly large number of cases of diaphragmatic hernias in adults have been discovered accidentally at autopsy and were entirely unsuspected during life. So long as strangulation does not occur the presence of the abdominal viscera within the chest is by no means incompatible with life. Ulceration of the stomach in its new situation is not uncommon, Koenboek¹¹ in 1914 reporting 32 such cases. These ulcers have occasionally ruptured and given rise to pyothorax, not, as would seem at first more likely, to peritonitis, the abdominal cavity being protected by the contraction of the diaphragmatic muscular ring about the opening. Most of the newborn cases with posterior defects and no sack die shortly after birth; those with a sack are more apt to live than those without, as shown by the small number of adults who show a posterior or central defect without a sack.

These hernias are coming to operation more and more frequently. In 1912 Scudder¹² could find only 53 cases which had been operated upon; since then there have been a number of cases successfully cured by surgery, those in which the herniation takes place through a dilated esophageal opening giving the most satisfactory results. The true hernias are easier to close than the false ones. Many long standing cases are of course incapable of reduction, either by the thoracic or abdominal approach, but much progress has been made since the days when metallic mercury was given by mouth for the purpose of reducing a herniated stomach by gravity.

EVENTRATION OF THE DIAPHRAGM.

This condition is not, strictly speaking, a true form of hernia of the diaphragm, but it has so long been considered as such in classifications of these hernias that it is justifiable to include it in the present discussion, the more so as its etiology

is, in part at least, congenital. It consists essentially in a great thinning out and weakening of the musculature of the diaphragm on one side, almost always the left, so that the dome rises high in the chest, often up to the level of the second rib. The lung above is compressed; the abdominal viscera rise to occupy the space below. It can readily be seen that a differential diagnosis between this condition and a true hernia with a large sack is often very difficult and one which has provoked an extensive discussion in the literature.

CLASSIFICATION.

Eventrations are best classified etiologically as suggested by Lerche:

1. Congenital.
2. Acquired (a) acute, (b) chronic.

The chronic cases are the uncured acute ones, the latter usually resulting from some pathologic condition, such as an acute infectious disease, affecting either the diaphragmatic muscle or the phrenic nerve. The question of etiology has given rise to a considerable difference of opinion and numerous arguments have been advanced in support of various theories.

Many authors claim that the majority of the cases are congenital, with the diaphragmatic muscle on one side atrophied at birth. This view is based: (1) on the occurrence of the condition in the newborn, in conjunction with other fetal abnormalities; (2) on the fact that most of the cases are left sided and in this respect resemble congenital hernia of the diaphragm; (3) on the absence of signs of pressure on the lungs, showing that the latter never attained full development, being small but normal in formation. On the other hand, opponents of this view cite the cases which have followed some definitely pathologic condition, either of a general nature, like typhoid fever, or local, as in empyema. Increased intra-abdominal pressure, spinal lesions involving the phrenic nerve and idiopathic muscular atrophy have also been assigned as causes. In all these it is of course difficult to disprove the contention that the eventration was present but unsuspected previous to the onset of the subsequent illness.

The bulk of the evidence seems to favor a congenital origin. In all lesions of the phrenic nerve, aside from local pathology as in one case of a suppurating lymph node, there is no reason why the left side should be so much more often affected than the right. The same holds true of general infectious diseases and muscular atrophies. The fact that the symmetry of the chest wall is not disturbed, except in cases of local thoracic suppuration, is another point in evidence of the fact that the eventration has been present from birth, since a later change in muscle tension on one side would show itself in an asymmetry of the chest wall.

If simple prolonged increase in intra-abdominal pressure were an important factor, then we should expect to find eventration more commonly in women than in men, whereas statistics show exactly the reverse. Failure of the lung to develop to its normal size and so to exert normal pressure against the diaphragm may allow a premature stretching of the developing muscle, though the latter condition may be the cause of failure in lung development. On the other hand, the almost universal finding of a dextrocardia suggests the mechanical effect of an acquired lesion, since congenital dextrocardia is rare and would be unlikely to accompany so large a proportion of diaphragmatic eventrations. They may both be congenital manifestations of a retarded development.

Microscopic examination of the diaphragm in these cases reveals an atrophy of the muscle fibers and also an aplasia of them. The phrenic nerve may also show a pathologic change, either secondary or primary, so that there may be an atrophy of the nerve with secondary aplasia of the muscle or vice versa or both.

It has been suggested by Weigert that an injury may occur to the phrenic nerve during birth, as in a brachial paralysis, but this fails to explain the frequency with which the left side is involved.

In 1915 Bayne-Jones¹³ collected 45 cases of eventration, most of which had been proved by autopsy. Of these, 8 were females, 37 males. The ages ranged from birth up to 75 years, but the majority were in adults. There were 7 cases associated with other congenital anomalies and 9 were classified as acquired, the rest as congenital.

We have collected below the cases reported since 1915. As a number of these have been proved by X-ray only, they are somewhat less authentic than those confirmed by autopsy or operation. This latter procedure has been attempted once with reported success, but eventration is generally considered to be a nonsurgical condition. It is certainly not comparable in this respect with diaphragmatic hernia. One case of spontaneous cure has been reported and confirmed by serial X-ray examinations.

It had been considered possible that this investigation of the diaphragm might throw some light on the question of cardiospasm and esophageal stricture. Beyond the suggestion that the former may be due to a spasm, not of the esophageal muscle but to that of an abnormally tonic or spastic diaphragm and the latter to the scar tissue following an unrecognized and healed rupture of the esophageal "opening," no further connection between these subjects has been observed.

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- A complete bibliography comprising about 300 references will be furnished on request.

TABULATION OF NONTRAUMATIC DIAPHRAGMATIC HERNIAS SINCE 1900 (Fig. 11)

No.	Year	Author	Sex	Age	Side	Type	Size (cm)	Position	Congenital or Acquired	Remarks
1	1904	Pianchu	M	Newborn	Left	False	2cm diam.	Central	Congenital	Diagnosis proved by X-ray.
2	1904	Pianchu	M	Newborn	Right	False	2cm diam.	Posterior	Congenital	X-ray only. Stomach not in chest.
3	1921	Hirsch	M	36	Left	True	?	?	Acquired	Ruptured stomach.
4	1905	Herz	M	30	Left	?	?	?	Congenital	
5	1906	Daxenberger	M	23	Left	False	2 small ones	One central near esophagus	Congenital plus pathologic rupture	Stomach in right posterior mediastinum.
6	1918	Bund	?	8 months	?	True	?	Dilated esophageal opening	Congenital	
7	1913	Rischbieth	?	Newborn	Left	False	?	Total left defect.	Congenital	
8	1913	Rischbieth	F	82	?	True	?	Dilated esophageal opening.	Acquired	Hernia passed up through ligamentum pulmonale.
9	1903	Liepmann	M	Newborn	Left	False	10x3	Posterior	Congenital	Cured by operation.
10	1920	Rowlands	M	19	Left	True	Small	Posterior	Acquired	
11	1915	Grutes	?	Newborn	Left	False	Small	Central	Congenital	
12	1914	Sküllern	M	55	Right	True	Small	Posterior	Acquired	Sack in folds of suspensory ligament of the liver.
13	1903	Lepage	?	Newborn	Right	False	Large	Parasternal	Congenital	Diagnosed by X-ray only.
14	1916	Freud	M	38	Right	True	?	Posterior	Acquired	Acute strangulation with death.
15	1910	Marie	F	28	Left	True	?	esophageal	Congenital	
16	1910	Dietler	F	16	Right	?	?	Posterior	?	Diagnosed by X-ray only.
17	1916	Vitrac	M	?	Left	False	5cm diam.	?	Congenital	Acute strangulation.
18	1917	Weinberger	M	31	Right	False	?	?	Congenital	Diagnosed by X-ray only.
19	1915	Monti	?	4 mos.	Left	?	?	?	Congenital	Diagnosed by X-ray only.
20	1921	Allard	M	?	Left	?	?	?	?	Diagnosed by X-ray only.
21	1916	Ruder	M	Newborn	Left	?	3cm diam.	?	Congenital	Foramen Bochdalecki distinct from defect.
22	1914	Gruher	M	48	Right	False	10cm diam.	Central	Congenital	Further herniation of stomach into right flank to ach into right chest to stimulate pycnophrosis.
23	1905	Hamdi	M	50	Right	True	3cm diam.	Posterior	Acquired	Many ulcers and adhesions.
24	1921	Jahn	F	Fetus	Left	False	3x5cm	?	Congenital	Secondary herniation from left to right chest through mediastinum.
25	1914	Cailloud	M	48	Right	?	Large	Central	Of Trauma ?	Liver and colon in right chest.
26	1911	Scholz	?	Newborn	Right	True	4cm diam.	?	Congenital	
27	1920	Seudder	M	16	Right	True	?	Para-esophageal	?	
28	1920	Huffman	F	Adult	?	True	?	Dilated esophageal opening	Congenital	Other hernias, abnormalities of blood vessels, only 11 ribs, patient for-oval.

TABULATION OF NON-TRAUMATIC DIAPHRAGMATIC

HERNIAS SINCE 1900 (Fig. 11)---Continued.

No.	Year	Author	Sex	Age	Side	Type	Size (cm)	Position	Congenital or Acquired	Remarks
29	1920	Mathews	F	Adult	?	True	?	Dilated esophageal opening	Congenital weak. Acq. hernia	Sack in right posterior mediastinum. Symptoms 10 years.
30	1919	DeCourcy	F	25	?	True	?	Dilated esophageal opening	?	Marked anemia. Operation and cure.
31	1919	Sorost	F	?	Left	?	?	Parasophageal Central	?	Operation. Symptoms only recently.
32	1912	Scudder	M	29	Left	False	?	No further data given	Congenital	
33-38	1903	Hertz	?	Newborn	Left	?	?		Congenital	Liver in chest.
39	1903	Hertz	F	Fetus	Right	False	Huge	Total half	Congenital	
40	1913	Potczki	?	Newborn	Right	False	Large	Posterior	Congenital	
41	1914	Gruber	M	9 days	Right	False	Moderate	Posterior	Congenital	Patent foramen ovale.
42	1914	Gruber	?	Newborn	Right	False	Large	Posterior	Congenital	Big liver. Common mesentery for small and large bowel.
43	1914	Gruber	F	Newborn	Left	False	Moderate	Posterior	Congenital	Myelocoele. Club foot. Entire mesentery free.
44	1914	Gruber	F	Fetus	Left	False	2cm diam.	Posterior	Congenital	Free bowel mesentery.
45	1914	Gruber	M	Fetus	Left	False	3cm diam.	Posterior	Congenital	Free mesentery.
46	1914	Gruber	F	Newborn	Left	False	Large	Posterior	Congenital	Free mesentery.
47	1914	Gruber	M	Newborn	Left	False	Large		Congenital	Accessory left lung. Free mesentery.
48	1914	Gruber	F	7 days	Left	False	2cm diam.	Posterior	Congenital	Free mesentery.
49	1914	Gruber	F	Fetus	Left	False	2cm diam.	Almost whole half	Congenital	Degenerate adrenal. Encephalomyelocoele. Free colonic mesentery.
50	1914	Gruber	M	Newborn	Left	False	Large	Whole half	Congenital	Persistent ductus arteriosus.
51	1921	Cohn	F	Newborn	Right	False	2cm diam.	Posterior	Congenital	Very large liver. Right lung embryonic.
52	1920	Mitchell	F	50	Left	True	15x20cm	Parasternal	?	Sack passed across midline to right chest.
53	1921	Bakes	M	43	Left	?	3x6cm	Posterior	?	Operation with freeing of phrenic nerve.
54	1905	Jonas	M	Newborn	Right	False	2cm diam.	?	Congenital	Liver very small.
55	1905		F	Newborn	Left	True	2x3cm	Central	Congenital	
56	1908	Don	M	61	Left	True	?	Dilated esophageal	?	
57	1912	Morestin	M	33	Left	False	?	Central	Congenital	Death due to intestinal obstruction. Operation followed by death. Type probably due to old trauma.
58	1903	De Cardinal	M	53	?	False	?	Pericardial	Acquired	None.

59	1907	Risel	F	Newborn	Left	False	4x4½cm	Posterior	Congenital	Portions of liver tissue in sack wall, showing liver to be a factor in its development.
60	1907	Risel	F	Newborn	Left	True	Large	Whole half	Congenital	Whole stomach in thorax with pylorus coming through esophageal opening. Stomach in posterior mediastinum.
61	1905	Chartier	?	4 mos.	?	True	Large	Dilated esophageal	Congenital	Anomalies of vessels.
62	1909	Bertier	F	Newborn	Left	False	?	Posterior	Congenital	Right lung underdeveloped.
63	1909	Bertier	F	Newborn	Right	True	?	Whole half	Congenital	Liver and gall bladder in right chest.
64	1906	Cantley	?	4 mos.	Right	True	?	?	Congenital	Heart grasped by hand. No symptoms.
65	1902	Moser	M	Adult	Left	True	?	Parasternal	?	Sack contained stomach, not liver.
66	1904		M	2 yrs.	Right	True	2x3cm	Posterior	Congenital	
67	1907	Hunter	?	Newborn	Right	False	?	?	Congenital	
68	1907	Hunter	?	Newborn	Left	False	?	?	Congenital	
69	1908	Williams	F	9 mos.	Left	False	?	Posterior	Congenital	Secondary herniation through posterior mediastinum.
70	1915	Kakels	M	54	Left	True	Large	Posterior	Congenital	No further data.
71	1921	Roos	M	35	Left	?	?	?	?	Patent foramen ovale and ductus arteriosus.
72	1918	Gross	?	Newborn	Left	True	No left muscle	Whole half	Congenital	Abnormal liver. Dextrocardia. Stenosis of aorta.
73	1918	Fisk	M	Newborn	Left	False	Large	Posterior	Congenital	Herniation of spleen through posterior mediastinum into right chest.
74	1913	Meyer	F	53	Left	True	7x6cm	Dilated esophageal	?	Strand of diaphragm across top of sack.
75	1908	Martland	M	70	Left	False	4x5cm	Pericardial	?	Transverse colon in pericardial sack; rest of diaphragm normal. No symptoms.
76	1910	Bokay	?	10 days	Left	True	4cm diam.	?	Congenital	
77	1910	Bokay	?	11 mos.	Left	True	?	Central	Congenital	Left lung size of raisin, kidneys embryonic.
78	1911	Rawes	?	?	Left	False	3cm diam.	?	?	
79	1914	Monks	M	43	Left	False	8cm diam.	?	Congenital	No symptoms.
80	1918	Dickie	?	Adult	?	True	?	Dilated esophageal	Acquired	
81	1920	Carmichael	?	Newborn	Left	?	?	Posterior	Congenital	Complicated by intestinal obstruction.
82	1921	Barnett	?	8 mos.	Left	True	Large	?	Congenital	Secondary mediastinal sack in which large bowel was strangulated.
83	1916	Gordon	M	56	Right	True	Large	Anterior	Acquired	Death from acute obstruction.
84	1913	Vogel	M	47	Left	False	15cm diam.	Posterior	Congenital	

No.	Year	Author	Sex	Age	Side	Type	Size (cm)	Position	Congenital or Acquired	Remarks
85	1907	Ulrich Walton	F	26	?	True	?	Dilated esophageal opening	?	Hernial sack in right chest.
86	1917	McClave	M	3½ mos.	Left	False	2cm diam.	Posterior	Congenital	Death.
87	1916	Miller	F	50	Left	False	?	Central	Congenital	Death due to obstruction.
88	1919	De Buys	?	4 mos.	Left	False	?	Posterior	Congenital	Death due to obstruction.
89	1914	Fletcher	?	2 yrs.	Left	?	2cm diam.	Central	Congenital	
90	1917	Bevan	F	41	?	True	?	Dilated esophageal opening	?	
91	1917	Bevan	F	45	?	True	?	Dilated esophageal opening	?	
92	1920	Mayer	?	Newborn	Left	False	2x5cm	Posterior	Congenital	Death from asphyxiation. Patent foramen ovale and ductus arteriosus. Rudimentary left lung. Left kidney in chest.
93	1921	Hajak	M	46	Left	True	5cm diam.	Posterior	Acquired	Entire left diaphragm muscle poor. Died of strangulation.
94	1919	Pohlman	M	63	Right	False	8cm diam.	Central	Congenital	Marked cirrhosis of liver.
95	1921	FitzMaurice Kelley	M	35	Both	True	?	Parasternal	Acquired	
96	1918	Shafer	?	2 mos.	Right	False	3cm diam.	Central	Congenital	
97	1917	Cockayne	?	Fetus	Left	False	2.5cm diam.	Posterior	Congenital	
98	1918	Lubosch	M	Adult	Both	True	1cm diam.	Parasternal	Acquired	Left accessory lungs (two). Accessory spleen.
99	1914	Rise	M	71	Left	False	2½x2cm	Posterior	Congenital	Sack in anterior mediastinum.
100	1918	Gross	M	53	Left	False	?	Anterior	?	Strangulation.
101	1903	Chadburn	M	30	Left	False	8x12cm	Central	Congenital	Death due to sudden gastric distension.
102	1907	Williams	M	77	Left	False	4cm diam.	Posterior	Congenital	No symptoms.
103	1911	Harris	F	31	Left	False	Whole half	?	Congenital	
104	1910	Ringrose	F	26	Left	False	8cm diam.	Posterior	Congenital	Died of obstruction following birth of child.
105	1922	Borden	F	25	Left	False	7cm diam.	Central	Congenital	Whooping cough a possible added factor.
106	1918	Downes	M	7	?	False	3cm	Dilated esophageal opening	?	
107	1909	Bechman	?	17	Left	False	?	?	Congenital	Reported as congenital but had a history of trauma.
108	1909	Bechman	?	47	Left	False	?	Posterior	Congenital	Symptoms for 18 months only.
109	1909	Bechman	?	47	?	True	?	Dilated esophageal opening	Acquired	
110	1920	Keith	?	17	Right	True	?	?	Congenital	
111	1917	Rautenberg	?	?	Left	?	?	Posterior	?	

112	1916	Seibert	F	68	?	True	?	Dilated esophageal opening	Acquired	History of three difficult labors. Viscera were in left thorax.
113	1915	Kilner	M	30	Right	?	?	Parasternal	Acquired	Gas between fold of falciform ligament which also contained the tip of right lung. Only case of thoracic viscera entering abdomen.
114	1919	Warren	?	16	?	True	?	Dilated esophageal opening	?	Hernia followed a left empyema two years before. Hernia diagnosed as ulcer and gastro-enterostomy performed. Hernia shown by X-ray three months later.
115	1919	Warren	?	30	?	True	?	Dilated esophageal opening	?	
116	1917	Kohler	F	12	Left	True	Entire half	?	Acquired	
117	1904	Taylor	F	Newborn	Left	True	3cm diam.	Posterior	Congenital	Huge liver. Duodenum with a free mesentery.
118	1904	Gilford	F	4 mos.	Right	False	?	Central	Congenital	
119	1904	Gilford	F	4 mos.	Right	False	?	Posterior	Congenital	
120	1921	Cleiz	F	Newborn	Left	False	?	Central	Congenital	
121	1913	Reiss	?	3 yrs.	Left	True	Large	Central	Congenital	
122	1920	Greive	F	5½ yrs.	?	True	5x8cm	Dilated esophageal opening	?	
123	1912	Waille	?	24	Both	True	?	Parasternal	?	
124	1920	Poster	M	24	Left	?	?	?	Acquired	Cough for 5 years. Repaired by operation. Both hernias repaired by operation.
125	1911	Mercade	M	27	Left	False	3cm diam.	Posterior	Congenital	Traumatic perforation of stomach in chest. Perforated stomach ulcer with pyothorax.
126	1911	Courmont	F	32	Left	?	5cm diam.	Central	?	Hernia followed a left empyema.
127	1908		M	7	Left	?	?	?	Acquired	
128	1904	Davergne	?	Newborn	Right	False	3cm diam.	Posterior	Congenital	
129	1912	Devraque	?	Newborn	Left	False	Huge	Posterior	Congenital	Other abnormalities including undescended testicle.
130	1911	Keck	M	Newborn	Left	True	3x4cm	?	Congenital	
131	1921	Bergman	M	Adult	?	True	?	Anterior epigastrium	Congenital & Acquired	Epigastric tumor with absence of lower sternum and anterior diaphragmatic muscle.
132	1908	Patel	M	69	Left	False	5x4cm	Anterior parasternal	?	Recent strangulation.
133	1909	Gret	M	58	Right	False	7x6cm	Anterior parasternal	?	Secondary protrusion of intercostal wall producing a visible tumor. Cardiac history with prolonged cough. Also an umbilical hernia.
134	1908	Berthaux	?	Newborn	Left	False	Huge	Posterior	Congenital	Abnormally large liver.
135	1908	Schreiber	F	Newborn	Left	False	Huge	Posterior	Congenital	Numerous other hernias in family, including father and mother.
136	1918	Du Pan	F	63	Left	True	3cm diam.	Central	Acquired	Previous repair of ventral hernia, a possible factor in increasing abdominal pressure.
137	1918	Montandos	M	41	?	True	?	Dilated esophageal opening	painboy	Severe strain a factor. Operation and death.

TABULATION OF CASES OF EVENTRATION SINCE 1915.

No.	Date	Author	Sex	Age	Side	Symptoms	Pathology Remarks
1	1920	Funk	F	39	L	Pain and vomiting.	Diagnosed by X-Ray.
2	1917	Neuman	M	44	L	History of pneumonia.	Atrophy of muscle and phrenic nerve.
3	1918	Aronson	F	20	R	History of asthma, pain in chest, vomiting.	Congenital megacolon; phrenic paralysis from trauma. Operation for megacolon.
4	1921	Korns	M	20	R	No history.	Aplasia of right lung, dextrocardia, hypospadias and undescended testicles.
5	1918	Assman	M	38	L	Sense of pressure in chest.	
6	1922	Langlen	F	10	L	Loss of weight.	
7	1920	Weigert		2 mo.	L	Case of high forceps.	Cured by operation with excision of centre of left diaphragm. Checked by X-Ray. ? of birth paralysis of phrenic nerve.
8	1919	Schwenke	M	23	L	Palpitation, pain in left side.	Diagnosed by X-Ray.
9	1919	Schwenke	M	30	L	Dysphagia.	Operation for hour glass stomach.
10	1917	Betchov	M	46	L	Cough, dyspnea.	Diagnosis by X-Ray.
11	1916	Wood		18			
12	1917	Minkowski	M	47	L	Vague symptoms for 2 years.	Severe trauma at seven years of age.
13							
14-20	1916	Weil				Five indefinite cases of left eventration without sufficient data to tabulate, and diagnosed by X-Ray only.	
21	1922	Lercher		37	L	Chronic cough.	Due to suppuration in lymph node involving phrenic nerve. Operation and cure.
				Total since 1915.....		21	
						Right	2
						Left	17
						Indef.	2

**TABULATION OF CASES REPORTED BY THOMA AND GROSSER
UP TO 1900.**

1. False Diaphragmatic Hernia (nontraumatic):			
a. Left—			
1. Total lack.....	27	(all congenital)	
2. Central defect.....	56	(29 adults, 5 children, 22 newborn)	
3. Posterior defect.....	17	(3 adults, 13 newborn, 1 child)	
b. Right—			
1. Total lack.....	5	(4 newborn, 1 child)	
2. Central defect.....	18	(8 adults, 2 children, 8 newborn)	
2. True Diaphragmatic Hernia (nontraumatic):			
a. Left—			
1. Anterior (parasternal).....	6	(all adult)	
2. Central	7	(6 adults, 1 newborn)	
3. Posterior	1	(1 child)	
b. Right—			
1. Anterior (parasternal).....	6	(all adult)	
2. Central	2	(both newborn)	
3. Posterior	1	(adult)	
c. Esophageal	4	(1 child, 3 newborn)	
3. Eventration:			
1. Left	7		
2. Right	1		
4. Location Indefinite:			
1. Left	61		
2. Side not mentioned.....	27		
5. Totals:			
1. Left	175	Adults	61
2. Right	42	Children	13
3. Eventration	8	Newborn	89
4. Esophageal	8		
True		27	
False		137	

STATISTICS ASSEMBLED FROM CASES SINCE 1900

Age	Newborn		Children		Adults	
Total	50		10		58	
Percentage	42.4%		8.6%		49%	
Sex	Male		Female			
Total	53		35			
Percentage	60%		40%			
Sack Present	Newborn		Children		Adults	
Total	9		3		31	
Percentage	18.3%		6.1%		63.5%	
42.2%					12.1%	
Sack Absent	Newborn		Children		Adults	
Total	38		3		22	
Percentage	56.7%		4.5%		32.8%	
57.8%					6.0%	
Adults	Sack		No Sack			
Total	31		22			
Percentage	58.5%		41.5%			
Children	Sack		No Sack			
Total	3		3			
Percentage	50%		50%			
Newborn	Sack		No Sack			
Total	9		38			
Percentage	19.1%		80.9%			
Location of Defect:	Left	Right	Anterior	Posterior	Central	Esophageal
Total	82	33	12	42	21	20
Percentage	71.3%	28.7%	12.4%	45.5%	22.1%	21%

(Exclusive of three cases of total lack of one-half the diaphragm.)

These statistics are necessarily somewhat inaccurate, since each item of data was not always given in each case, but they serve to show the relative frequency with which these hernias occur, both as regards their situation and their type.



FIGURE 1.

Diagram illustrating formation of the heart in the guinea pig. The splanchnic mesoderm over the vitelline veins is migrating medially to fuse with the same layer on the other side.

(After Strahl and Carius.)

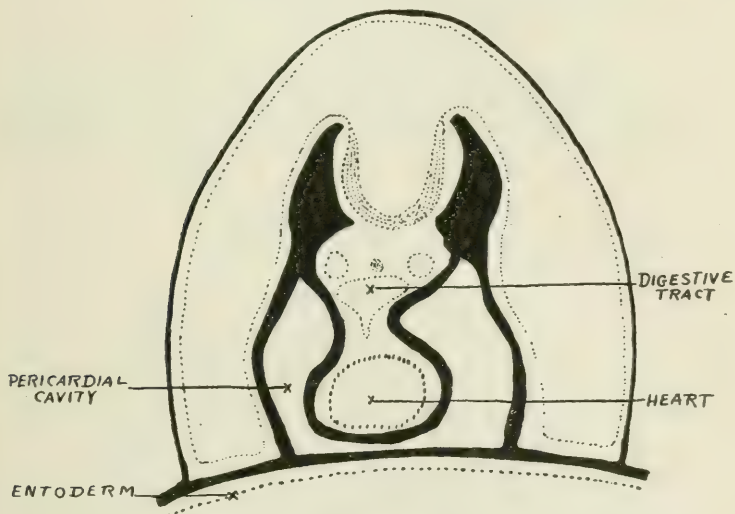


FIGURE 2.

Later stage of Fig. 1. The two vitelline veins have fused to form the heart and the fusion of the splanchnic mesoderm of each side has formed the pericardial cavity. (After Strahl and Carius.)

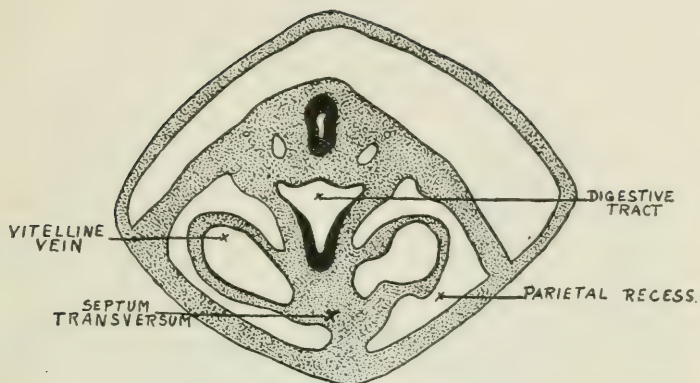


FIGURE 3.

Diagram to illustrate the formation of the lateral portions of the septum transversum by the outgrowth of the vitelline veins toward the body wall and into the parietal recesses. (Cross section of rabbit after Ravn.)

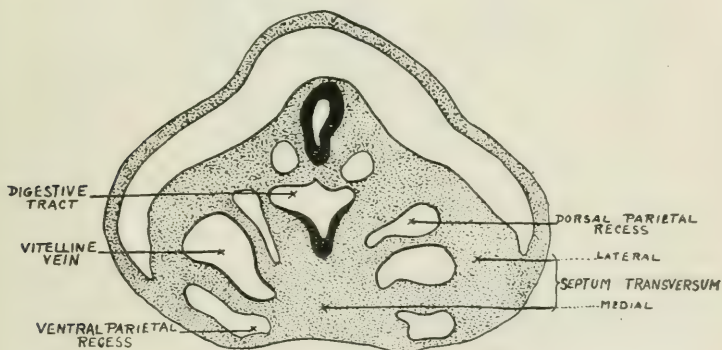


FIGURE 4.

Later stage of Fig. 1. The vitelline veins have fused with the body wall, forming the lateral portions of the septum transversum and dividing the parietal recesses into dorsal and ventral recesses. (Ravn.)

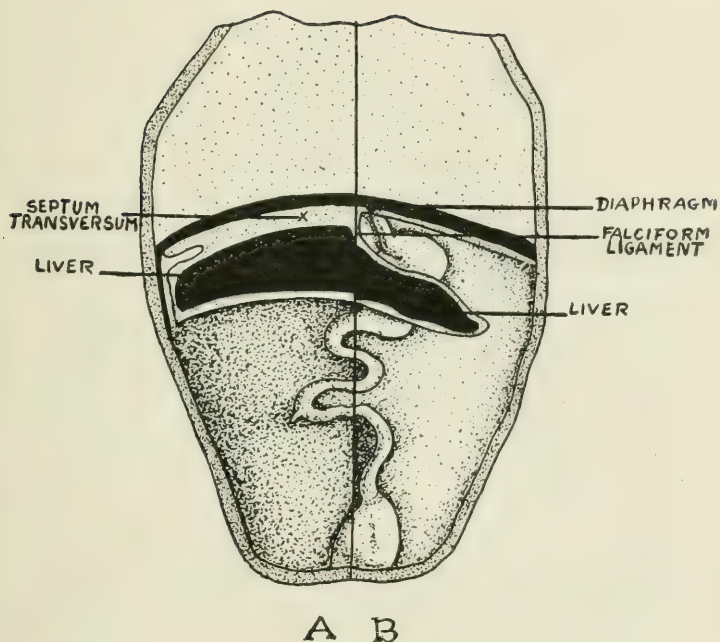


FIGURE 5.

Diagram showing the liver enclosed within the transverse septum and the manner in which infolding of the peritoneum constricts off the liver and forms the diaphragm and falciform ligament.

A.—Early stage. B.—Later stage. (Modified after McMurich.)

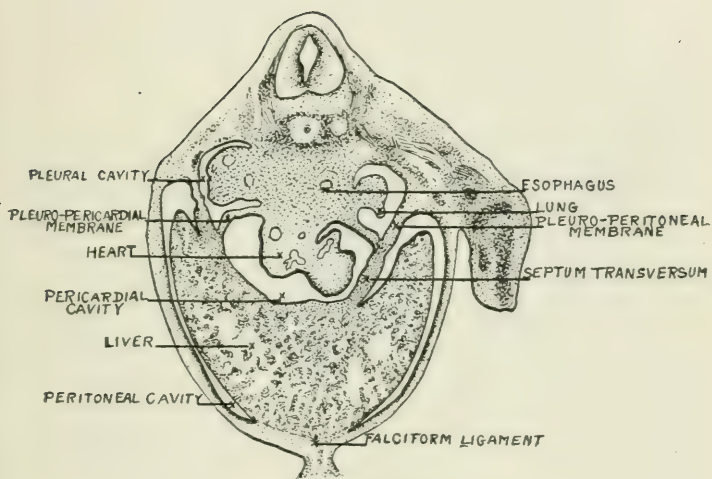


FIGURE 6.

Transverse section of a 10 mm. human embryo showing the pleuropericardial and pleuroperitoneal membranes separating the pericardial, pleural and peritoneal cavities. (After Prentiss.)

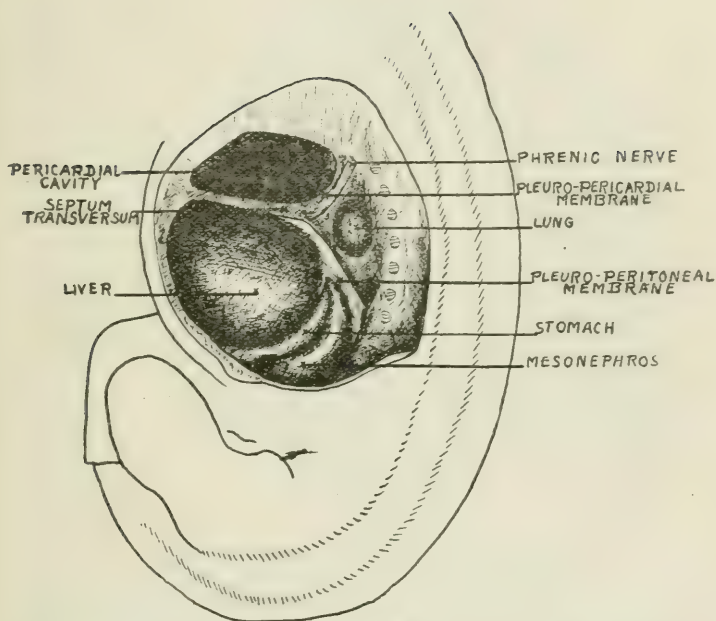
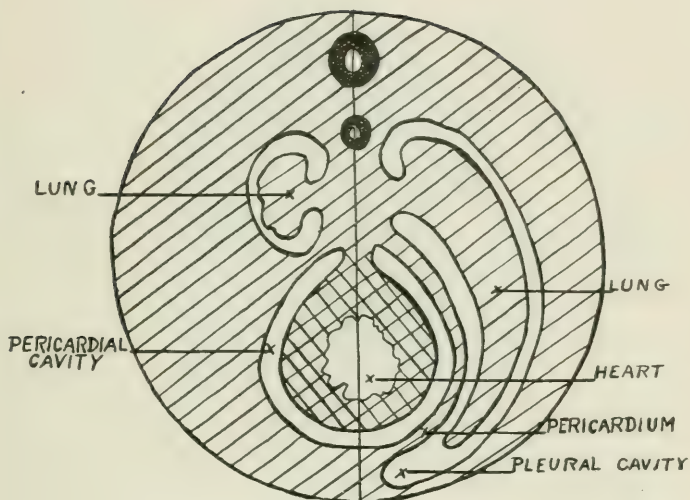


FIGURE 7.

Reconstruction of the 11 mm. embryo to show the relations of the pericardium, septum transversum, lungs and liver and their separation by the pleuropericardial and pleuroperitoneal membranes. (After Mall.)



AB

FIGURE 8.

Diagram to show the pericardium and its formation by the forward extension of the lung.

A.—Early stage. B.—Later stage. (Modified after Robinson and Prentiss.)

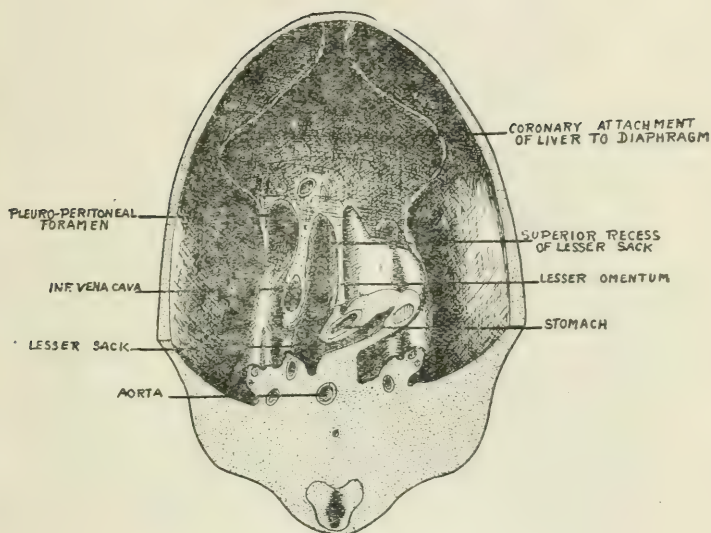


FIGURE 9.

Diagrammatic ventral view of middle third of 12 mm. embryo. The figure shows the upward extension of the lesser sack parallel to the lower end of the esophagus. The liver has been removed from its attachment to the diaphragm. The communication between the pleural and peritoneal cavities via the pleuroperitoneal canals is still open. (Based on figures of Mall and F. T. Lewis, and model by H. C. Tracy.)

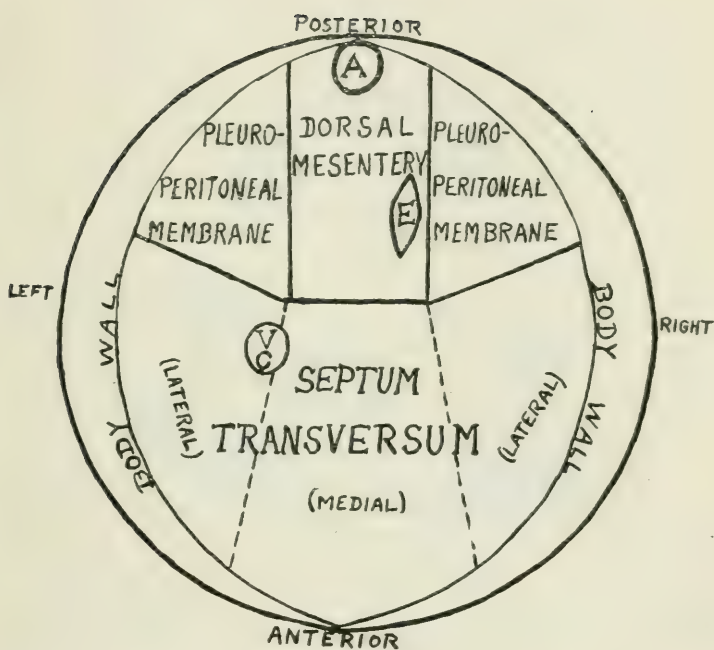


FIGURE 10.

Diagram to show the constituent portions of the adult diaphragm and the relative position occupied by them at an early embryological stage.

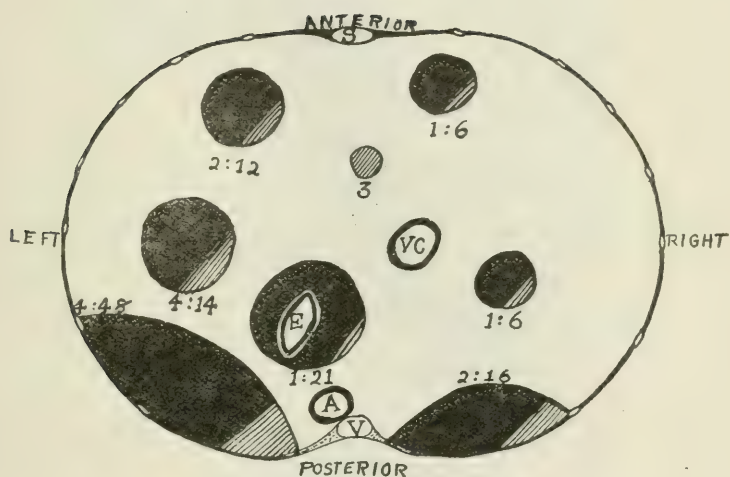


FIGURE 11.

Diagram to illustrate the positions and types of nontraumatic diaphragmatic hernia reported since 1900. The figures represent the number of cases in each location, the false hernias being shown in solid black, the true ones cross hatched.

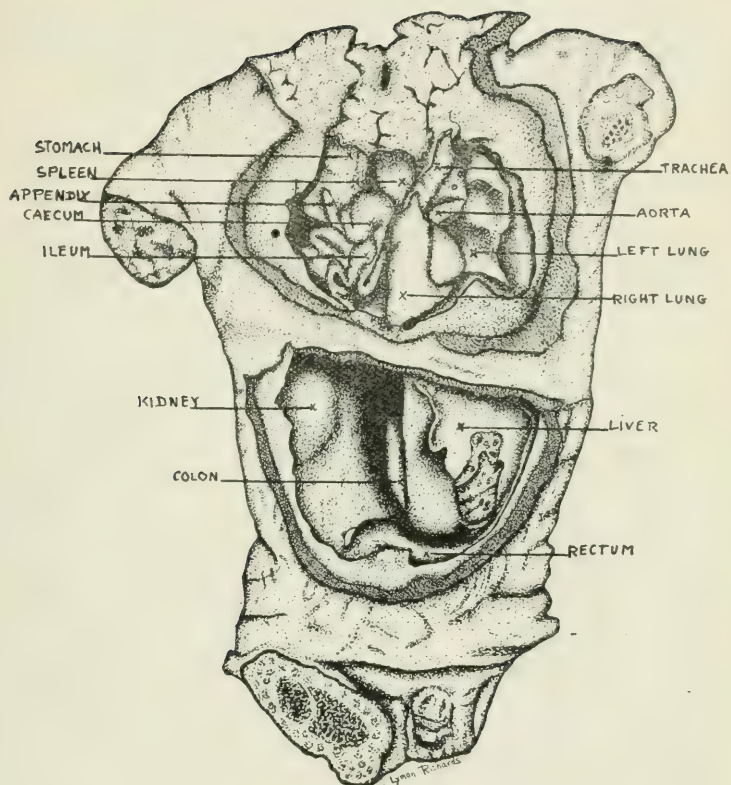
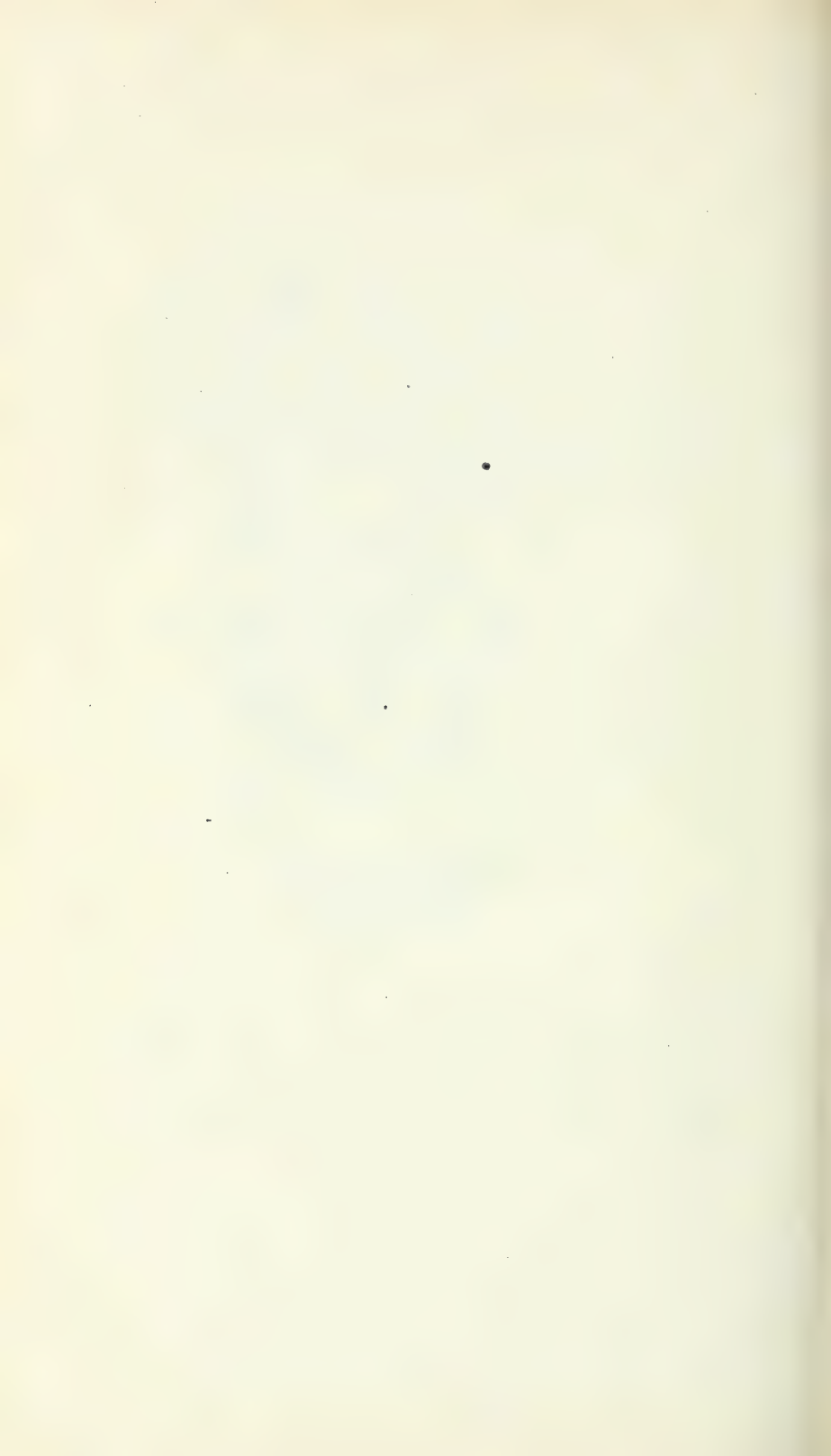


FIGURE 12.

Torso of male fetus, showing an extensive esophageal hernia of the diaphragm. The stomach, spleen, small intestine, and a large part of the large intestine have become lodged in the right thorax. There is apparently no sack, an unusual condition in this type of hernia. The heart has been removed, also the right half of the liver, showing the relatively great size of the left half. (From the collection of Dr. Harris P. Mosher, Harvard Medical School.)



LXXIX.

RADICAL OPERATIONS ON THE MAXILLARY
SINUS AND DAMAGE TO THE TEETH.

BY ROBERT H. IVY, M. D., D. D. S.,

PHILADELPHIA.

It has long been recognized that for the cure of the majority of cases of chronic inflammation of the maxillary sinus nothing short of a radical operation will suffice. It is conceded that nasal puncture and lavage will clear up most of the acute cases, but where the mucous membrane has become chronically thickened and the seat of polypoid degeneration, with or without bone necrosis, mere washing out through needle or canula from the nose has little effect. Again, this measure, while in the presence of free pus it generally establishes a diagnosis of maxillary sinus disease, may entirely fail to do so when the disease is manifested chiefly by the presence of solid granulation tissue. Establishment of free intranasal drainage by formation of a large window in the antranasal wall may occasionally be sufficient. However, in long standing cases, nothing is nearly so satisfactory for exact diagnosis and treatment as exposure of the interior of the sinus to direct inspection and palpation through a large opening from the mouth through the buccal wall. This permits removal of the diseased tissue with a curette, the operation being completed by establishment of permanent nasal drainage into the inferior meatus through a resection of the nasal wall and final closure of the original buccal opening. Objections have been advanced against these radical operations that they frequently result in permanent damage to healthy teeth in the vicinity of the buccal incision. Thus, J. L. Myers (*Jour. A. M. A.*, 1921, lxxvii, 2081) calls attention to the possibility of devitalization of teeth by interference with their blood and nerve supply. At the suggestion of your Secretary I have endeavored to gather a few facts to determine whether or not there is any foundation for these objections.

Damage to sound teeth might conceivably be brought about in two or three ways: (1) By exposure of roots of teeth by removal of bone covering them; (2) By destruction of their vascular supply; (3) By destruction of their nerve supply.

1.—Exposure of Roots of Teeth by Removal of Bone Covering Them.—It is a generally known fact that if the supporting alveolar process and pericemental membrane about a tooth are destroyed by injury or disease, they will never be replaced, a focus of infection will usually be set up and the root will eventually be lost. It is conceivable that in doing the Caldwell-Luc or the Denker operation, if the incision through the buccal plate be made too low, the roots of some of the teeth near the apices might be exposed and thus converted into necrotic foreign bodies from loss of nourishing and supporting tissue. The nerve and blood supply of the pulp might also be cut off. Any such tooth injury would probably interfere with the success of the antrum operation, by setting up a new focus of infection. This accident is avoided by making the buccal incision well above the apices of the roots and being careful not to remove the bone too low down. None of the cases examined by me postoperatively showed either clinical or radiographic evidence that any injury to the teeth had occurred in this manner. Of course, these remarks do not apply to diseased teeth having a direct connection with the sinus prior to the operation, and which were removed at the operation.

2.—Damage by Destruction of Vascular or Nerve Supply of the Teeth or Both.—The arterial and nerve supply to the upper teeth, alveolar process and mucoperiosteum of the maxillary sinus reach these parts through minute canals in the substance of the bone and grooves in the wall and floor of the maxillary sinus. The posterior superior dental or alveolar artery and nerve enter the tuberosity of the maxilla on its posterior surface and supply more especially the molar teeth and surrounding alveolar process and gum, also the posterior part of the mucoperiosteum of the maxillary sinus. The canine and incisor teeth are supplied by the anterior dental artery and nerve which are given off in the infraorbital canal just before the infraorbital artery and nerve make their exit through the infraorbital foramen. They pass down canals in the anterior wall of the maxillary sinus to go to the teeth mentioned as

well as to the gums, alveolar process and mucoperiosteum of the anterior part of the sinus. The anterior and posterior blood vessels form a very rich anastomosis in the floor of the maxillary sinus, so that it is inconceivable that any individual tooth could have its vitality impaired by cutting a dental arterial branch unless immediately at the apex of the root of the tooth. There is usually a middle dental nerve branch from the infraorbital canal which supplies more particularly the region of the premolar teeth. While the dental nerve branches also form a very rich interlacing plexus in the floor and outer wall of the maxillary sinus, there is, of course, no true anastomosis of nerve fibers and the zones of distribution are more clearly defined than in the case of the blood vessels. While some of the blood vessels must necessarily be cut in radical operations on the maxillary sinus, the rich anastomosis will in all cases insure a sufficient collateral circulation to maintain the blood supply of the tooth pulps. Interference with blood supply by these operations, then, need not be regarded as a factor in injury to the teeth.

Removal of a large portion of the outer bony and membranous wall of the maxillary sinus of necessity produces a resection of many of these nerve fibers to the teeth. In the Denker operation, where the bone removal is carried right through to the anterior nares, a large section of the anterior dental nerve is inevitably destroyed, with resultant loss of sensation in the incisor and canine teeth. Does mere loss of sensation mean that the pulps of these teeth are devitalized? I think not. One can have anesthesia of the fingers due to sensory nerve lesion, without gangrene. Vitality depends on presence of blood supply and not on sensory nerve supply. Absence of sensation in a tooth does not necessarily mean that the pulp is dead, although death of the pulp of a tooth following inflammation is accompanied by loss of sensation. Where a tooth pulp dies from inflammation, decomposition will frequently produce a change from the normal color, and such a tooth will appear darker than its fellows with vital pulps. These changes are not seen in teeth whose nerve supply has been interfered with at such a distance that the blood supply is not also cut off, as in fractures of the mandible or in radical maxillary sinus operations.

A postoperative study of the upper teeth was made in a series of 20 cases which had been subjected to radical maxillary sinus operations, either Denker or Caldwell-Luc type, at periods ranging from three months to two years after the operation. As far as possible, it was determined by questioning the patient whether any teeth on the side of the jaw involved had been the seat of postoperative disturbance. Practically all patients noticed a numbness in the teeth and gums beneath the buccal incision, which lasted only a few weeks or months in some, in others for a year or more. The apparently sound teeth on the side of operation were then tested for pulp vitality by means of the faradic current. Where crowned or obviously pulpless teeth were present careful inquiry was made to determine whether devitalization had occurred prior to or after the antrum operation. When teeth did not respond to the faradic test or were obviously pulpless, radiographic films were made for evidence of periapical bone or root injury produced in the operation.

In the cases examined shortly after operation, the faradic test frequently showed absent or diminished reaction. This was particularly true in the canine and incisor teeth of cases subjected to the Denker operation, where the anterior dental nerve was removed. In practically all cases tested several months after operation absolutely normal faradic reaction was observed in the teeth, indicating regeneration of the lost segment of nerve.

In only one case was there any history of trouble in a tooth after the operation. In this case the lateral incisor had been treated for pericementitis due to a dead pulp, the radiograph showing an area of bone destruction about the apex. Even here it is by no means certain that this pulp was alive before the operation. In no case was there any radiographic evidence that injury to the periapical bone tissue or to the roots themselves had occurred at the time of operation.

While the series of cases is not very large, as a result of these examinations, I believe we are justified in concluding that fears of deleterious effects to the teeth from radical maxillary sinus operations of the Caldwell-Luc and Denker types, when performed by experienced surgeons, are entirely unfounded. Furthermore, if occasional damage to a tooth did

result, this should not be used as an argument to condemn a type of operation which is the only satisfactory curative procedure in many cases of chronic maxillary sinusitis.

Grateful acknowledgment is here made to Drs. R. H. Skillern, G. M. Coates, Ralph Butler, B. D. Parish, and others, whose cases in addition to those of the author furnished the data for this report.

SUMMARY OF CASES.

Operation.	Examination of Teeth.	Notes.
	Nov. 2, 1922.	2nd premolar nonvital prior to operation.
1. I. M. Sept. 21, '21. R. Caldwell-Luc.	+ + - m + - + 7 6 5 4 3 2 1	Pulp of lateral incisor probably became devitalized after operation, with area of periapical infection shown by X-ray.
	April 5, 1923.	No abnormality found.
2. C. B. Dec. 10, '21. R. Denker.	+ + m + + + + 7 6 5 4 3 2 1	Teeth felt numb for a time after operation, but this gradually passed off.
	April 5, 1923.	Normal sensation in all teeth except 2nd premolar, which was pulpless before operation.
3. W. W. May 8, '22. L. Denker.	+ + + + - m + 1 2 3 4 5 6 7	
	March 20, 1923.	Sensation still slightly diminished in incisors and canine. 2nd molar nonvital before operation.
4. E. B. Mar., 1920. R. Denker.	- m + + + + + 7 6 5 4 3 2 1	
	Nov. 1, 1922.	Sensation the same as before operation.
5. M. M. Nov., 1921. R. Side.	m m m + m + + 7 6 5 4 3 2 1	
	Nov. 1, 1922.	Numbness of incisor and canine teeth one week after operation.
6. M. M. Oct. 23, '22. L. Side.	- - - + m m m 1 2 3 4 5 6 7	
	Nov. 18, 1922.	Diminished reaction in incisors and canine. Premolars devitalized before operation.
7. J. S. June 2, '22. R. Side.	+ m - - + + + 7 6 5 4 3 2 1	
	Nov. 18, 1923.	Complete return of sensation.
8. W. B. Mar., 1919. L. Side.	+ + + + m + + 1 2 3 4 5 6 7	
	Nov. 16, 1922.	Complete return of sensation.
9. C. B. May 13, '22. L. Caldwell-Luc.	+ + + + + + + 1 2 3 4 5 6 7	

Operation.	Examination of Teeth.	Notes.
	March 22, 1923.	Complete return of sensation. Crowned teeth normal.
10. H. S. Dec. 10, '21. L. Denker.	+ + + c m m c 1 2 3 4 5 6 7	
	March 22, 1923.	Complete return of normal sensation.
11. L. F. Nov., 1922. L. Denker.	+ + + + + + + 1 2 3 4 5 6 7	
	April 3, 1923.	Bridgework was on before operation. No change found, except slightly diminished sensation in central incisor.
12. M. E. Feb., 1921. L. Side.	Bridge + m — m c + + 1 2 3 4 5 6 7	
	March 8, 1923.	Negative reaction in lateral incisor, no X-ray abnormality.
13. E. G. Sept., 1922. L. Caldwell-Luc.	+ — + + m + + 1 2 3 4 5 6 7	
	March 8, 1923.	Still has numb feeling on right side, though teeth respond to current.
14. E. G. Dec., 1922. L. Caldwell-Luc.	m m m + + + + 7 6 5 4 3 2 1	
	March 28, 1923.	Still has diminished reaction in 1st premolar. 1st molar devitalized before operation.
15. J. M. Dec., 1922. L. Caldwell-Luc.	+ — + + + + + 7 6 5 4 3 2 1	
	March 23, 1923.	Numbness gradually passing off, except in 1st premolar. No other abnormality found in this tooth.
16. H. D. Nov. 1, '22. L. Caldwell-Luc.	+ + + — + m + 1 2 3 4 5 6 7	
	April 7, 1923.	All teeth responded to test before operation. Sensation has not yet returned to certain teeth, but they show no other abnormalities.
17. S. D. Jan., 1923. R. Caldwell-Luc.	+ + — + — — — 7 6 5 4 3 2 1	
	April 9, 1923.	Numbness still present in lateral incisor and 2nd premolar.
18. J. G. Jan., 1923. L. Side.	+ — + + — + + 1 2 3 4 5 6 7	
	April 6, 1923.	Sensation normal in central incisor.
19. F. B. Feb. 8, '23. L. Denker.	+ m m m m m m 1 2 3 4 5 6 7	
	April 21, 1923.	Complete return of sensation in incisors. Canine and 2nd molar now used to support bridge.
20. W. N. Feb., 1922. R. Denker.	c m m m c + + 7 6 5 4 3 2 1	

Explanation of notation: 1, central incisor; 2, lateral incisor; 3, canine; 4, 1st premolar; 5, 2nd premolar; 6, 1st molar; 7, 2nd molar; plus sign, presence of sensation; minus sign, absence of sensation; c, crown; m, missing.

LXXX.

OTITIC CHOLESTEATOMATA.*

BY S. MACCUEEN SMITH, M. D.,

PHILADELPHIA.

It is not my purpose to review extensively the literature of otitic cholesteatoma, my chief object being to present an unusual case of extradural tumor of this nature; neither shall I consider the various well recognized methods, other than surgical, of treating aural suppuration, whether cholesteatoma be present or not. The object of this paper is to bring forth a discussion of the etiology, histology and pathology of otitic cholesteatoma, its surgical removal and the prevention of its recurrence in so far as possible. It is interesting to note that the more recent views differ but little from those of the masters of twenty or thirty years ago; indeed, the whole present conception of this peculiar formation is based on the contributions of an earlier period.

With the object of determining whether cholesteatomata are found in osseous structures other than the temporal bone, their legitimate and historic habitat, I have inquired of a number of general surgeons, including J. Chalmers DaCosta, John B. Deaver and Charles H. Frazier, all of whom gave a negative answer, the notable exceptions being Harvey Cushing¹ and Percival Bailey,² but the tumors which they report may differ, however, in some important aspects from the otitic cholesteatomata long since recognized by aurists as a consequence of chronic otorrhea. We all recognize the migratory propensity of cholesteatomata and their ability to destroy the soft tissue and penetrate the osseous structure; it would not be unreasonable, therefore, to assume that growths of this character adjacent to the organ of hearing had their origin in the temporal bone, even though no active process in the latter was immediately discoverable.

*Read before the annual meeting of the American Otological Society, Atlantic City, May, 1923.

Cases are reported in which cholesteatomata have been found in the eye, brain, spinal cord and kidneys. It is possible also that these might differ in some essential respects from the type under consideration.

Should an aural discharge, however innocuous, continue for a long period, necrotic destruction of the connective tissue elements ensues. In the presence of a diminishing suppuration, new connective tissue formations are induced, which serve to protect the underlying osseous structure, so that it frequently resists the ravages of the various microorganisms and the necrotic changes incident thereto for an indefinite time. Sooner or later, however, the normal blood supply and the nutrient vessels are interfered with, and those parts least supplied, such as the incus, first succumb.

Once the soft parts have been destroyed, the tympanic and its accessory cavities are wholly deprived of their every means of resistance to the devastation of invading bacteria, and thus originate caries and necrosis and the nidus for the development of cholesteatoma.

When the pneumatic cells of the mastoid process become obliterated in the course of a chronic proliferative osteitis, acute exacerbations are more conducive to intracranial complications through caries of the tympanic roof or the posterior wall of the mastoid, exposing the dura or lateral sinus.

Gruber and other early authors believed these cholesteatomatous formations to be malignant in character, and seemingly substantiated their conclusions by minute examinations of the masses. More recent views consider them primarily benign, and if ever malignant they become so in the evolution through which they pass. It is also shown that these masses invade the osseous structure through the Haversian canals, even in the early stages of erosion and before caries and necrosis supervene, which explains the tendency to recurrence after thorough extirpation seems to have been attained.

These masses, composed of concentrically arranged epithelium, cholesterol crystals and inflammatory débris, literally swarm with microorganisms. Their gradual growth or enlargement first destroys the soft parts and then produces dilatation of the bony cavities, more especially the mastoid antrum, and this in turn is followed by pressure necrosis of the under-

lying osseous structure, frequently exposing and subsequently involving the dura, lateral sinus, and rarely even the brain itself in an acute process of extremely dangerous potentiality.

Cholesteatomata vary in size from that of a small pea to a walnut. They are usually round, but their final contour is governed somewhat by the shape of the cavity in which they form; however, when the bone undergoes necrotic change the mass may penetrate it and mould its outline according to the resistance encountered.

Except for occasional mild attacks of giddiness and headache and a feeling of weight and pressure in the head, cholesteatomata may remain in the temporal bone for years without causing symptoms. Curiously enough, cases of this kind do not seem to be recognized as of sufficient importance to interest the patient and sometimes not even the physician. Several of my recent cases did not seek relief until they had had severe attacks of vertigo, even to the extent of necessitating an attendant. Others suddenly became almost totally deaf or developed Bell's palsy before they were impressed with the gravity of the situation. Although these patients are aware of the existence of a chronic ear disease, a fair percentage insist that they cannot remember how long ago an actual discharge was present, this in spite of the fact that an examination reveals an otorrhea, frequently very offensive.

Under the stimulus of an exacerbation, the mass, wherever situated, takes on acute inflammatory changes which lead to extensive destruction of tissue through necrosis. In their development, cholesteatomata of the tympanic cavity may follow the line of least resistance and extend well into the external auditory canal; on the other hand, cases have been reported as penetrating the eustachian tube. The latter I have never observed, but I have seen them through a carious erosion of the mastoid cortex. Likewise, they may cause labyrinthitis, meningitis, sinus thrombosis or brain abscess.

A unique case which I have hitherto hesitated to report, but which will be detailed later on, deals with a large cholesteatoma which invaded the temporal lobe, being thoroughly walled off and enclosed in a capsule, perhaps spurious in type, the patient presenting no symptoms, either local or general, indicating its presence.

I have never seen what may be termed true cholesteatoma except that which developed in the course of or as a result of chronic otitic suppuration, and, furthermore, my observation leads me to believe that the tympanic and accessory cavities, together with the marginal perforations of the membrana tympani, are alone the site of these strange formations. Some are reported occupying the external auditory canal, but when it is remembered that the meatal epidermis does not proliferate pathologically, and thus give rise to the pearly white dermic scales and other characteristics until it reaches the tympanum and adjacent cavities, it is reasonable to assume that these recorded meatal cases are not of the true cholesteatomatous type. Formations of cholesteatoma are, of course, found in the deep external canal, but are associated with and are actually an outgrowth from the process in the middle ear cavity, or protrude through a fistula leading into the attic or antrum.

Those who have seen a considerable number of cholesteatomatous cases must have been impressed with the following facts: That they are observed only in the presence of chronic suppuration; that their formation is inhibited when the otorrhea is profuse; that they are practically never found in central perforations of the membrana tympani unless the margin is partially adherent to the tympanic wall; and that their development nearly always occurs in chronic suppuration of the attic or perforations of the superior and posterior quadrant. This would seem to support the generally accepted theory that the primary stage of their evolution is the ingrowth of epithelium through the marginal perforations; their subsequent well known characteristics being developed by the histologic changes through which they pass.

Another distinctive feature of cholesteatoma is the temporary decrease of suppuration, almost to the point of cessation in some instances, when the mass has attained a considerable size. This is the type of case that may be quiescent until some moisture or infection invades the ear, producing recurrent attacks of mastoiditis, when they swell hygroscopically, as is shown by headache, vomiting, vertigo, facial palsy, etc., ushering in the well recognized and dangerous acute exacerbations. It is in this recurrent type of case that I have observed the great majority of intracranial complications.

Lucien-Barajas³ has clearly shown that there are two formations found in the middle ear under the name of cholesteatoma. One is composed of granular, fatty and purulent masses of detritus, intermixed with squamous epithelial cells, keratin and crystals of cholesterin, while the other variety is formed by imbricated nodules concentrically arranged like the layers of an onion. These peculiar nodules consist microscopically of large fat, endothelial cells, polygonal in shape, the nuclei staining but faintly, while the masses contain a large quantity of cholesterin. The major portion of our cases belong undoubtedly to the first group and must not be classed as true cholesteatoma, nor should similar masses be placed in this class when composed of polystratifications of keratinic epithelial cells, which develop inflammatory dermal affections of the external auditory canal and which are horny or keratinic formations.

Personally I doubt the existence of the so-called primary otitic cholesteatomata. To be such they must necessarily originate independently of tympanic suppuration, and therefore not pass through the characteristic evolution. This would mean either that they are not cholesteatomata or else the tympanic perforation may have temporarily closed over the underlying mass, of which this tumor was originally an outgrowth. Ballenger,⁴ speaking of secondary cholesteatoma, says this is the type found in practice; the primary form is chiefly limited to literature.

Meatal epidermal layers may find their way into the tympanic cavity during the course of a receding chronic suppuration, and thus provide the necessary epidermal lining to produce the so-called spontaneous cure of a chronic suppurative otitis media. It will be seen, therefore, that the mere presence of epidermal scales in a chronic middle ear suppuration does not in any sense constitute or portray the distinctive features of cholesteatoma. Epidermal layers are an integral part and essential to ultimate completeness of the finished product, but this end is not accomplished until the underlying osseous structure becomes involved, and the perforating epidermis degenerates into excessive pathologic proliferation and the course of evolution through which it passes is completed.

To some extent hyperplasia of the epithelium occurs in all cases of chronic otitic suppuration, but these formations cannot acquire the typical cholesteatomatous state until they are excessive and have remained amid the environment of the middle ear, attic or antrum sufficiently long to pass through the distinctive cholesteatomatous evolution.

The admittance of moisture in some instances causes decomposition of the mass, which is followed by the formation of granulation tissue or even polypoid growths, and this in turn favors the spreading of septic material to the meninges, brain and blood vessels through the osseous deficiencies incident to the changes resulting from pressure necrosis, the extent of which is dependent almost wholly on the size of the ever increasing mass.

Granulation tissue or polypoid growths originating in Shrapnell's membrane, more particularly in the superior posterior quadrant, are, in my experience, almost always the outgrowth of cholesteatomatous masses occupying the attic and sometimes the antrum also. When the lower part of the membrana tympani is intact, the characteristic sound produced by inflation is not heard, owing to the fact that the mass in the upper part of the cavity seals off the opening in Shrapnell's membrane. In such instances it is not uncommon for the incus, the head of the malleus and their ligaments to be absorbed through pressure necrosis.

Pressure atrophy and consequent necrosis due to cholesteatoma have been the principal underlying factors in virtually all of the cases of the so-called nature's radical mastoid operation that I have observed. On the other hand, I recall several patients in whom the cavities of the antrum and middle ear were wholly exposed, through nature's operation, and yet were both dry and empty.

Cholesteatomata not only offer a most favorable breeding ground for the rapid multiplication of organisms, but the very nature of their component parts serves to protect the germs and the new pabulum as it is secreted against our efforts at extinction by means of antiseptics, germicides and so on. Our chief reliance, therefore, as an initial step for relief lies in surgical intervention.

The cholesteatoma described by Virchow, a heteroplastic new growth of congenital origin, must be differentiated from that long since recognized by aurists, which is always secondary to a chronic otorrhea. I doubt very much whether a suppurative otitis media is ever caused primarily by cholesteatoma, as intimated by Harvey Cushing;⁵ if so, then our conceptions of the etiology and histology of otitic cholesteatoma must undergo complete revision.

I have read with much interest the informative contributions of Harvey Cushing⁶ and Percival Bailey⁷ referring to extradural cholesteatomata. After reviewing several cases by various authors, Cushing states that "these cases constitute a group of true epidermal cholesteatomata which are extradural and appear to originate between the two tables of the skull. The tables become separated, thinned by pressure and finally absorbed, the inner table as a rule suffering more than the outer. . . . Unquestionably the removal of the epidermal membrane is the crux of the procedure, and this can only be accomplished before the cyst is emptied of its contents and its enveloping membrane permitted to collapse. Incomplete removal of the lining wall is doubtless the explanation of the recurrences so common after operation by otologists for the cholesteatomata of the temporal bone which come almost solely into their province."

From the fact that otitic cholesteatoma does not usually develop until the soft parts have been mostly destroyed and the bone involved at least in a simple erosion, it is evident that aurists do not encounter the distinctive "lining wall" referred to by Cushing, and furthermore, it seems to be generally conceded that recurrences in otitic cases, to which he alludes, are the result of a subsequent outgrowth of the mass which has penetrated the Haversian canals and not to incomplete removal of an assumed "lining wall."

In operating, it is of course our object to remove as thoroughly as possible all new connective tissue, as well as other inflammatory debris, and I feel that we accomplish our object reasonably well, especially when due consideration is given to the dangers and difficulties encountered in the field of operation, which includes the thin plates of bone covering the fallopian canal conveying the tympanic branch of the facial nerve,

the carotid artery, the jugular bulb, various parts of the labyrinth and the lateral sinus.

Bailey⁸ intimates that Cruveilhier's "pearly tumors" are never found in the temporal bone complicating a chronic suppurative otitis media. It may be true that otitic cholesteatomata do not contain all of the characteristics of this classic type, as demonstrated by laboratory findings, and yet from a clinical standpoint some answer to this description in almost every detail. After all, may it not be a reasonable assumption that these extradural cholesteatomata had their origin in a former otitic disease of the type that assumed a spontaneous cure? If otology is to be deprived of the distinctive position it has maintained relative to the etiology and histology of otitic cholesteatomata, it will at least require much additional research and clinical observation before surrender of our seemingly well established facts will be acknowledged.

In order to secure permanent relief, cholesteatomata must be removed in their entirety so far as this is possible. Even then, if the suppuration continues, the irritation resulting is apt to cause recurrence, and when the suppuration ceases continued desquamation and proliferation are very apt to bring about the same result. It is evident, therefore, that the two objects to be attained after removal of the mass are cessation of the discharge and cessation or limitation of pathologic proliferation. I believe that we have at our command corrective agencies to accomplish both of these results in a favorable percentage of cases. Skin grafting, especially as practiced by Dench, after Ballance, seems to give very satisfactory results. For those of us who are not so skilled in this procedure, packing the cavity with small strips of gauze thoroughly moistened with a 1/1000 or 1/500 solution of acriflavin (the former strength being mostly used), will prevent the recurrence of cholesteatomata in many cases, even of a most obstinate type. This packing must be renewed at first every day, then every third or fourth day, until all evidence of further discharge or proliferation has ceased. Should there be evidence of recurrence afterwards, I have found a liberal application of a 1 per cent or stronger solution of mercurochrome to be quite efficacious—in fact, at this stage mercurochrome gives even better results than a continuation of the acriflavin.

After all, we must consider the complete eradication of cholesteatoma as not only difficult, but in some cases perhaps impossible of accomplishment, from the fact that even in slightly eroded bone the mass will force its way into the Haversian canals, forming focal centers from which it may develop in the future.

I have been using acriflavin for about three years in the treatment of cholesteatoma and mercurochrome for a somewhat shorter time, and although sufficient time has not elapsed for me to make a definite statement as to the permanent benefit thus derived, this treatment does seem not only to inhibit the formation of cholesteatoma, but acts as a specific against recurrence after thorough removal.

A sane rule to follow is that when a diagnosis of cholesteatoma has been made, or even when there is a strong probability of its existence, surgical intervention should be recommended if the case does not yield promptly to nonsurgical treatment.

When there is sufficient reason to believe that the process is limited to the tympanic cavity, operation through the external canal is justified. This operation necessarily includes removal of any granulation tissue and polypi that so frequently accompany cholesteatoma, as well as excision of the fragments of the membrana tympani, malleus and incus. Following removal of the mass, and ossiculectomy, packing with acriflavin solution, 1/1000, followed by the use of mercurochrome, as above described, should be carried out carefully to correct the otorrhea and prevent the return of pathologic proliferation.

The report of the case I referred to above follows:

J. W., male, aged 33 years, locomotive engineer. Well nourished, apparently enjoying best of health, very alert mind. Good habits and denied venereal infection. Suppurative otitis media, recurrent in type, extending over a number of years. Six years ago otorrhea ceased for four years, although patient admitted "a slight moisture in the ear" at times in the interval. Two years prior to operation an acute exacerbation followed the entrance of water into the ear, causing earache, pain in head, vertigo and emesis for a brief time. Discharge of varying quantity was constant from this date until relieved by operation. Latterly the discharge was scanty, brownish yel-

low, blood streaked and offensive. Patient stated that his ear was "punctured," whatever this procedure may have been, at onset of exacerbation, and the discharge continued to be sanguinolent during the intervening two years. Patient was free from all general symptoms but sought relief from an annoying discharge.

Left ear normal. Membrana tympani of right ear largely destroyed. Granulation tissue and what seemed to be a cholesteatomatous mass protruded through a large opening in the superior posterior quadrant and the necrotic opening in the adjacent posterior osseous wall. No ossicles observable. Nothing abnormal revealed by ocular examination. Chest normal with the exception of a few rales, the result of a recent cold. Abdominal examination negative. Blood examination and urinalysis normal.

A radical mastoid operation was performed. Cortex extremely hard and thick. Cells below level of antrum, having undergone sclerotic changes, were filled in with solid ivory-like bone. Antrum of normal size, filled with a cholesteatomatous mass, some granulation tissue and a very little free pus. Through the destroyed tegmen antri and tympani this cholesteatomatous mass pushed its way into the interior of the skull, forcing the dura before it through the temporal region to a depth of about $1\frac{3}{4}$ inches.

Before reading Cushing's instructive monograph⁹, wherein he reports a cholesteatoma the size of an adult fist, weighing 175 grams, four inches in diameter, and not producing any general symptoms indicative of its presence, I hesitated to report this case, as I could not conceive that the dura could be reflected to such an extent without causing some symptoms.

The opening through the tegmen antri and tympani was much smaller than the cavity beyond, so that the mass ballooned considerably at its distal boundary. This cholesteatomatous mass was first removed from the antrum and then from the ballooned intracranial cavity. On account of the possibility of inducing a meningitis in a healthy, ambulatory case, no attempt was made to remove the growth en masse, so I can only say that the tumor approximated in size a small pear. It had every characteristic of a typical otitic cholesteatoma.

The walls of the resulting cavity pulsated noticeably on removal of the mass.

The cavity was packed with a 1/1000 acriflavin solution, this being changed every day. The patient made an uneventful recovery and is now following his usual occupation.

As this was the first growth of this type that I had ever seen involving the interior of the skull, I asked my colleague, Dr. J. Clarence Keeler, who assisted in the operation, to furnish me his own estimate of its position and size. This he gave me as follows:

"The cavity, which was filled with cholesteatoma, appeared to be in the brain tissue, walled off by a thin layer of connective tissue. The walls pulsated after the cavity was evacuated. Location and size of cavity measured by memory on an adult temporal bone: Cavity was above the tentorium and extended along the anterior bar of the petrous bone to the extent of $1\frac{1}{2}$ inches. About $\frac{5}{16}$ of an inch of the brain cortex formed the outer wall lying next to the internal surface of the squamous plate, which did not appear to be involved. The upper depth of the cavity was about $\frac{3}{4}$ of an inch. The cavity extended upward, forward and inward as given per measurements, with a width of about $\frac{5}{8}$ of an inch."

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LXXXI.

A SURVEY OF THE HAY FEVER QUESTION—A
CRITICAL REVIEW OF THE SITUATION
RELATIVE TO THE ETIOLOGY
AND TREATMENT.*

BY OTTO J. STEIN, M. D.,

CHICAGO.

In order to discuss certain phases of the hay fever question it will be necessary to make at least a cursory survey of the subject in general, including, with the seasonal pollen disease, several of the vasomotor types of rhinitis.

The name "hay fever" has frequently been applied to most any form of vasomotor disturbance of the nose, although improperly so. The term itself does not correctly express the full meaning of the disorder. The acceptance of pollen causation should firmly establish its nomenclature, and the name pollen disease, or perhaps pollinosis as suggested by Todd, should preferably be used. Although Walker still speaks of a "perennial hay fever due to animal emanations and foods." (Jour. A. M. A., September 18, 1920, and ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1922.)

In the past hundred years many theories have been advanced as to the cause, and as many different names have been used. Beschorner in 1886 found in the literature fifty or more names for the disorder. This was because the nasal symptoms of pollen disease are the same as those of a multiplicity of other causes. Many of the theories that were then advanced are inapplicable to the present conception of this disorder. Some of them are true pollen disease of seasonal character and respond to the cutaneous test, while others are a type of hyperesthetic rhinitis of diverse etiology, sometimes being mechanical, bacterial or of fish, fowl or animal origin. A logical no-

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menclature would be pollen disease due to pollen causation and hyperesthetic rhinitis, a vasomotor nasal disturbance of manifold etiology.

From the time Bostock gave the first concise description of the disorder in 1819, under the name *catarrhus aestivus*, down to Dunbar's publications in 1902 and 1903, many theories prevailed as to the cause. These various causes were supposed to affect the nose: First, from without, as from solar heat rays, light rays, fine dust, odors, essential oils, chemical fumes and animal substance. Second, from within the nose, as synechia, rhinolith, septal ridge and spur, hyperesthetic areas and polyps. This view was sponsored by Daly in 1881 and enthusiastically supported by Roe, Sajous, Hack, Hertzog, Bosworth and others. Third, from a perverted state of the body fluids, chiefly an excess of uric acid in the blood creating a condition called uricemia. The champions of this theory were Molliere, 1884; Leflaine, 1887; Cheatham, 1890; Leseur, 1895; Haig and Bishop in 1894.

The theory of neurosis and psychic disorder was stoutly supported by John C. Mackenzie and followers in 1884. Some went so far as to class them ironically either as "neurotic, erotic or tommyrotic." After this came the theory of infection from Helmholtz, Heyman and Sticker. In 1900 Holbrook Curtis enthusiastically sponsored a preparation made from common ragweed, under the name of liquor ambrosia, while Ingals experimented with a liquid made from ragweed and goldenrod.

For a time interest in the subject lagged, then suddenly Dunbar created local interest by the presentation of his extensive investigations, wherein an albuminous principle obtained from pollen of certain cereals and grasses was isolated and to which was ascribed toxic action. At first only passive concern was aroused, even though Elliotson in 1831, Majendie in 1839, Abbott-Smith in 1865 and Blackley in 1872 had presented ideas somewhat similar. They ascribed the irritation of the pollen to a mechanical action of its spines and prickles and not to a protein of a toxalbuminous nature as Dunbar did. This toxic principle, extracted from wheat and rye and later goldenrod, was injected into young horses, after trying goats and rabbits, and he obtained a serum of antitoxic action. Ow-

ing to its unstable nature, it was mixed with an inert powder and sold under the name of pollantin. MacCoy referred to this as the first instance of the production of antitoxic serum where the animal and vegetable product have been crossed. He obtained complete and prompt relief in fifteen cases. Somers made a serum from animals inoculated with goldenrod pollen and reported prompt and positive amelioration of symptoms in a majority of ten cases with a smaller number of complete disappearance. Mohr, Joachim, Loeb, Stein, Semon, Curtis and many others used it with varying results, in the main successful as to the amelioration of symptoms and a shortened time. Its disuse was occasioned partly because of the necessity for a special serum from each pollen cause. After Dunbar awakened interest in the theory of toxicity from plant pollens and also in his passive immunity with serums, attention was directed to other proteins, such as animal hair and skin, feathers of birds and the proteins from foods and bacteria. Clark and Meyer in a recent article report a unique case of susceptibility to silk from the glue used in its manufacture. The result of these numerous observations brought forth a long list of new causes added to the list of known vegetable like pollens, so that the determination of the particular offending protein often assumes the proportion of a huge task. In order to learn the nature of an individual's susceptibility, a skin test may be made, using such proteins as may suggest themselves from a careful history of a given case. A certain amount of skill is necessary in its performance. At least care in its performance and caution in its interpretation are demanded. Skin tests may be misleading and not infrequently reveal multiple causes. The younger the age, the more sensitive the test. Where the cause, for instance, is found to be a food, or a toilet article, or wearing apparel, its elimination may suffice in bringing relief. A change in geographic locality may remove the offending pollen. These are simple instances of simplified treatment. After having determined the offending protein by allergic reaction or otherwise, the next natural step was to prepare a vaccine that would desensitize the individual. As has been the case in the past with other forms of treatment, much enthusiasm has been aroused. Many able and conscientious workers have contrib-

uted to the large volume of literature on the subject, and distinct progress is noted of late in improved technic. Conspicuous in this field of endeavor are the names of Noon and Freeman in 1911, who were pioneers in demonstrating active immunity, Vaughan, Abderhalden, Clowes, Koessler, Goettlieb, Coco, George Mackenzie and Walker. Their writings summed up indicate that in applying treatment for active immunization particular attention should be given to the following points: 1. A careful study of each case to ascertain its specific protein or proteins. 2. All contributing causes, constitutional or local, must be corrected. 3. The proper preparation and preservation of the vaccine. 4. The time of commencing treatment. This must be early enough to permit the development of a gradual immunity, about three months prior to the expected attack. 5. The time to stop treatment. Even though pre-seasonal treatment has been taken, severe anaphylactic shock may result from a sudden exhibition of quantities of protein, such as may occur at the height of the flowering season. Garnet (*Jour. A. M. A.*, September, 1920) anticipates this by applying adrenalin to the nose at this time. In the case of food proteins it may also be necessary to withdraw the faulty food. 6. Exact dosage, commencing with minimum strength and amount and very gradually increasing. According to Walker (*ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, September, 1922), a 0.1 or 0.2 c. c. of that strength solution should at first be used, which just failed to give any skin reaction. 7. Interval between treatments. Usually five to seven days.

Failure to observe these general rules, through ignorance or carelessness, may result in serious, if not fatal, consequences. Bessau says: "Active immunization against hay fever is not entirely free from harm and must be practiced with greater caution." He reports a case of a female, 20 years old, who, after the seventh vaccination in fifteen months, complained of a feeling of contraction within the nose, face and throat; sneezing, vertigo, great weakness and prostration. Cyanosis, edema, particularly of the nose and ears, dyspnea, mucus in the air passages, weak compressible pulse, cold hands. After two hours, urticarial rash, towards evening many symptoms still present. During the night very restless, mouth

breathing and an attack of sneezing after midnight, twenty odd times, with lacrimation, itching and so forth, typical hay fever symptoms, which disappeared in half an hour. During the following day some of the symptoms still persisted. Dunbar, Walters and others have recorded similar experiences with pollen poisoning. Ersner raises the question whether hay fever and asthma are due to a deficient protective substance in the body or the result of overstimulation with toxins. He believes overstimulation is the answer, and to administer pollen during the acute stage of hay fever is adding a torch to the fire already burning. Patients thus treated become worse, and no pollen treatment, therefore, should be given at this time. He tries to develop an immunity by beginning his injections at least three months before the time of the attack, and then starts the injection of stock vaccine against the rhinitis that accompanies the hay fever. Most of the authorities (Gottlieb, Sheppegrell, Selfridge and Walker) insist on the individual pollen treatment determined by previous skin tests. Others still use mixed pollens. If of the same biologic type this would seem permissible, although evidence has shown otherwise at times. To carry out a consistent pollen treatment, recognition must be given to those pollens in a given geographic area, even if immunity is obtained from related botanical groups (Goodale). No immunity results from unrelated groups (Clowes). Hence the treatment of the East is not necessarily the remedy for the West, and this is also true of the North and the South, Europe and America.

There is an economic side to this question that is never commented on by the physician, but very much so by the patient, namely, the cost of the treatment in time consumed and services rendered.

The pathology of bacterial disease causing hyperesthetic nasal symptoms is generally understood and accepted. The pathogenicity of pollen disease is less understood. In fact, it seems to be the weakest link in the chain. There must be an altered state of the chemistry of the body fluids which creates the individual's susceptibility. That here lies the fundamental cause of the disorder only a heretic can deny, and much hope is placed in the investigation that physiologic chemistry must soon reveal. Majendie (1839) and later Wolff-Eisner

suggested a protein sensitization by parenteral digestion as the underlying cause of the reaction. Koessler (*Journal of Infectious Diseases*, 1914) emphasizes this when he says "the proteolytic enzyme of the nasal secretions splits the foreign pollen gradually into harmless proteoses and aminoacids," and the small amount of contained toxin is so slowly and in such minute amounts absorbed as to be negligible in effect. A sudden and large amount of toxin resorption injures the epithelial cells, so as to permit easy permeability of the proteins. There also results a local tissue sensitization through the production of a proteolytic ferment which later becomes generalized throughout the body.

Inseparable from this thought is that of the relationship of internal secretions. It is understood that not only the endocrine glands but most every tissue of the body contributes an internal secretion. Accurate knowledge as to whether these hormones influence the tissues of the body so as to be a determining factor in the creation of a state of susceptibility, will be awaited with interest. Until such proof is presented reliance for the present must rest upon a considerable clinical observation. A long list of writers have directed attention to such a relationship, notably John Mackenzie, Solis Cohen, Bates, Pottenger, I. Chandler Walker, Sajous, Zubelin and Grant Selfridge. The beneficial results obtained from the employment of thyroid and pituitary extract, particularly in children, is worthy of attention; also the universal use of adrenalin in asthma and vasomotor rhinitis, suggesting a gland dyscrasia. If sensitization to a given protein cannot be demonstrated, the treatment by gland extract often brings about the desired relief.

Looking at the subject as a nasal reflex there is sufficient anatomic and physiologic information to account for the various vasomotor symptoms. The immense area of peripheral exposure offered by the membranes of the respiratory passages and conjunctival surface favors a degree of irritation and absorption present in no other part of the body. These membranes are supplied with a nerve mechanism of such complexity that the various impressions can be carried to all parts of the body. The nerve fibers that emerge from the sphenomaxillary fossa and that are distributed to the nasal and acces-

sory nasal membranes contain sensory, secretomotor, vasodilator and vasoconstrictor fibers. Brubaker (Jour. A. M. A., August 23, 1919) states that the effects of a local disturbance of the nose may by afferent pathways so enormously stimulate the associated central cells as to allow an overflow of impulse into neighboring vasodilator, secretomotor and visceromotor centers in the medulla and cord as to cause respiratory, cutaneous, circulatory and visceral symptoms. Sluder (Jour. A. M. A., August 23, 1919), quoting Langley, accounts for the nasal reflex origin of asthma by the impulses from the nose, passing from the nasal ganglion to the vidian, carotid plexus and lower cervical ganglion and thence by the way of the thoracic nerves to the lung. The work of Blos, Yonge, May, Gordon and my own was principally based on the previous knowledge that experimental stimulation of Meckel's ganglion or the peripheral cut end of the great petrosal caused hyperemia of the nasal membrane with dilatation of vessels and great increase of secretion. Upon this information has been built a theory of inhibition, which put into practice brought about a cessation of the reflex phenomena. In this is found a satisfactory explanation of the beneficial results obtained in these cases from the injection of alcohol into the nerve supply areas of the nose. If the nerves are properly blocked at the various exits in the nose there will result an inhibition of the various sensory sympathetic symptoms with a corresponding cessation of the disorder. The *modus operandi* of this form of treatment I have presented at various times.

Now, as to the results obtained by the various methods. There is no accurate way of obtaining reliable information. The best that can be done is to rely upon the statement of the patient. This may be correct and it may be incorrect. From the use of pollen vaccine treatment, from preparations furnished by commercial houses, the information furnished on actual results cannot be reliable, because these reports come from all over the country, from many physicians, used under varying conditions. In many instances the cases have not been carefully selected nor properly studied or followed up and accurate results in some are not known. The observations of Walker (ANNALS OF OTOLGY, RHINOLOGY AND LARYN-

GOLOGY, No. 3, 1922), in the preseasonal treatment of 115 grass cases showed about 50 per cent free or practically free from symptoms, and of 420 ragweed pollen cases about 40 per cent free or practically free. The results obtained from treatment during the attack were not as good: 32 per cent practically free in the grass cases, and 14 per cent free or practically free in the ragweed cases. This represents an average result obtained by many others. Besredka and G. M. Mackenzie have shown the value of local desensitization by the application of the antigen to the nasal and respiratory membranes, Parks favoring ragweed pollen for this purpose. Caulfield combines the subcutaneous with the local application.

The bacterial vaccines, especially when used in conjunction with methods tending to eradicate active foci of infection, offer many instances of cures. Even removal of the infectious process without the added vaccine has brought the relief sought for. The work of Schadle, Fink and Helmholz have proven this.

The serum treatment of Dunbar and his followers in this country (Somers, Cohen, Joachim, Loeb, Stein) have shown beneficial results that compare favorably in many instances with those of the vaccine treatment of today.

Mention might be made of the results obtained by Henske with the use of autogenous defibrinated blood. He obtained permanent relief in 56 per cent and temporary relief in 25 per cent of 16 cases of bronchial asthma and hay fever.

With the alcohol treatment injected locally in the nose just prior to the expected attack the beneficial results in 100 cases show 80 per cent free or almost free from symptoms.

This percentage can be improved by more careful selection of cases and a more accurate technic than is always used. It has the advantage of requiring only one or two treatments and therefore, as an economic factor, it has every advantage.

CONCLUSIONS.

Notable progress has been made in the pathogenesis of this disorder.

From the evidence at hand the opinion prevails that the disorder is one of sensitization.

That the antecedent cause of this state of sensitization is as yet not fully understood, but probably there is a particular

altered state of the fluids of the body. Whether this is a chemical change in the elements or simply a disproportion of substances or an excess or lack of some endocrin secretion, the future of physiologic chemistry must determine.

That hay fever symptoms may be due to an anaphylactic reaction or a reflex action.

That many different external causes, acting as the local exciting irritant exist, and in the sensitive create symptoms of hyperesthetic rhinitis. Without both factors actively being present at the same time no symptoms can result.

The use of specific pollen solution, when properly selected, prepared and administered, will materially relieve a small number of hay fever patients. Immunity when obtained is usually temporary, and when so, treatment must be repeated as often as is required. The unskilled administration, as well as the employment of improperly prepared or unstable serologic products may contribute to the sensitization of an individual. Even in the hands of the skilled, alarming and serious results have followed the use of vaccines. The percentage of cures obtained from the employment of pollen vaccines is not greater than results obtained by other methods.

That the results obtained from the nerve blocking method with alcohol give the greatest percentage of freedom of symptoms. That this method of treatment can be given at a much less expense of time and money to the patient. That the possibility of unfavorable results is minimum.

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LXXXII.

BRAIN ABSCESS OF OTITIC ORIGIN IN THE LEFT TEMPORO-OCCIPITAL REGION WITH ALL THE TYPICAL SYMPTOMS. OPERATION. RECOVERY.

BY ALEXANDER ZEBROWSKI, M. D.,
NEW YORK.

G. S., 50 years of age, complains of severe pains in the left ear and in the left mastoid region, accompanied by fever and otorrhea. All these symptoms appeared suddenly three days ago. However, the otorrhea had persisted for about 20 years but was not constant. The patient says that during the last five years the left ear was always dry. Status praesens: A tall, well nourished man; in the lungs, heart and abdomen nothing abnormal. The right ear, nose and retropharyngeal cavity normal.

The left ear: Voice, 5; whisper, 2. Weber lateralized to the left ear. Rinne negative. Fistula test negative. Profuse purulent discharge.

Otoscopic examination: Drum membrane, red in color and swollen. There is a small round shaped perforation in the front of the mallet. The superioposterior section of the drum is bulging (Fig. 1). Temperature, 103 F.; pulse rate, 116.

On the same day paracentesis of the left drum membrane was made, the incision being through the whole length of the drum.

On the next day the pain in the ear disappeared. Temperature, 100 F. Slight tenderness over the right mastoid region only on pressure. After application of the usual treatment the general condition of the patient improved markedly. The purulent discharge became scanty, the mastoid process painless, temperature normal. Suddenly, ten days after the paracentesis, the patient experienced a terrible headache, confined exclusively to the left parietal region. Simultaneously he had nausea and vomiting. Temperature, 102 F.; pulse rate, 72.

The examination of the ear revealed nothing suspicious.

The old perforation was of the same size as previously, a thin scar after the incision being noticed and a scanty mucopurulent discharge is present. The mastoid process was not painful or sensitive, and the hearing on the left side was slightly diminished. Caloric reaction normal. The rise of the temperature and the intense headache lasted for three days. At the same time I noticed some slight changes in the behavior of the patient. Usually rather talkative and disposed to complain of his sufferings, he gradually became taciturn, answered the questions after some hesitation and complained of headache, loss of appetite and sleeplessness. The mastoid process was absolutely not sensitive. Temperature and pulse rate normal. Three and one-half weeks after the purulent discharge appeared, the headache suddenly disappeared and at the same time the patient showed symptoms of amnesic aphasia. Changing from his taciturn disposition, he quite suddenly began to discuss questions which had nothing to do with his illness, using queer expressions and wrong words. The patient stammered, used unrecognizable words and forgot the names of many things, such as "handkerchief," "penknife," "key." It was impossible to state if the alexia and agraphia were present, because the patient constantly refused to write or to read anything. The pulse rate was markedly retarded, 54. Temperature at night, 98 F. The examination of the eye by an ophthalmologist revealed on the left side a marked choked disc, on the right side dilated blood vessels.

Next day a modified radical mastoid operation was performed. After the typical incision, the periosteum was found not thickened and not adherent, the mastoid process totally sclerotic, the antrum of a normal size containing a slight quantity of pus. The roof of the antrum had a normal appearance, no fistula being visible. Careful probing of the tympanic cavity revealed quite smooth surface of the ossicles, no granulations, therefore the ossicles were left in situ. The posteriosuperior part of the external bony meatus was removed up to the attachment of the drum. In the direction of the posterior cranial fossa we met some mastoid cells filled with the pus. The surrounding bone was hard as ivory. The posterior cranial fossa was opened and the wall of the sinus found healthy. The middle cranial fossa was opened, the dura show-

ing a normal appearance. Two punctures anteriorly and two straight were without result. Incision with a scalpel to the depth of $3\frac{1}{2}$ cm. also revealed no pus. The fifth puncture far posteriorly towards the occipital lobe revealed an abscess at the depth of 4 cm. I got in the syringe more than 8 cc. of pus, grayish in color and odorless. The free incision through the dura and the brain tissue evacuated about two tablespoons of pus. Drainage of the abscess cavity by means of iodoform gauze. Panse's plastic. The bacteriologic examination of the pus showed that the infection was produced by the streptococcus pyogenes, pure culture of the streptococcus being found with no mixed infection.

Next day the patient had some headache and the general condition improved. Temperature, 94 F.; the pulse rate, 74 to 68. The third day after the operation dressings were changed. About one tablespoon of a pus, creamy in color and odorless, was evacuated under pressure. The after treatment consisted in the change of the dressing at first every day, afterwards every second day. On the 16th day after the operation the opening in the dura was closed. The after treatment of the wound after the radical operation lasted about three months. The patient, however, returned to his usual occupation four weeks after the operation. The ear was dry.

The symptoms of aphasia and disartria disappeared suddenly on the fourth day after the operation. On this day the patient read the papers and wrote a letter. The retardation of the pulse disappeared immediately after the evacuation of the abscess. On the operating table the pulse rate ran from 54 to 74, and even to 80. The general condition and the appearance of the patient were greatly improved; while in the hospital he gained nine pounds in three weeks. The examination of the eyes showed disc normal. The hearing was fairly good, 5 for the whisper. Behind the auricle a lineal scar was to be seen.

Epicrisis.—The case described is, as I have already stated, a classic example of an otitic brain abscess developed suddenly during a chronic otorrhea. Being under observation from the very beginning of the complication allowed us to state how long it took for the development of the abscess. It is very likely that this time was at any rate shorter than four weeks,

because the first symptoms appeared earlier than a month after the previously dry ear began to discharge. Such a short space of time has already been described, by Jansen in his textbook, a case of brain abscess, the patient being 69 years old, in which a rupture into the lateral ventricle occurred 14 days after the previously healthy ear began to discharge.

The disturbances of speech, disartria and aphasia appeared very early and were markedly developed. The operation revealed that the abscess was located far away from the center of speech—it was found in the posterior part of the left temporal lobe in the occipital region. The aphasia was probably due to the collateral edema and encephalitis. After the evacuation of the pus the speech of the patient was normal on the fourth day. In this case the disturbances of speech—usually the most important symptom for the localization of a left sided brain abscess—did not assist in locating the abscess. It was found only after four unsuccessful punctures and not in the place where presumably it should be found. In the cases of otitic left sided brain abscess which I have previously described (*Monatschrift f. Ohrenheilkunde*, 1905 and 1911), the abscess was always found very near to the center of speech.

In this case the infection of the brain was due to the streptococcus pyogenes. In the article of Blau*, a comparative study of 1,000 cases of endocranial complications of chronic purulent otitis media, in 218 cases where a bacteriologic examination was made, in 68 the infection was due to the streptococcus pyogenes. In my case it was practically a pure culture of these bacteria, no mixed infection being present. This indicates that the abscess was recent.

The otoscopic picture in my case was quite unusual, because in the monograph of Blau cited above we do not find an analogous case. According to Blau, the endocranial complications are nearly always accompanied by the large or total destruction of the drum.

Among the 1,000 cases of Blau there were 234 brain abscesses. The eyes were examined in 135 cases—the choked

*Louis Blau: *Zur Lehre von otogenen intrakraniellen Erkrankungen*. Beiträge zur Anatomie, Physiologie, Pathologie und Therapie des Ohres, der Nase und des Halses, 1917, page 86.

disc was found only in 14 per cent of the cases. In 46 per cent of the cases the disc was normal, in 40 per cent the changes were but slight, dilated blood vessels or optic neuritis.

It is interesting to trace the way through which the infection reached the posterior cranial fossa. In this case the infection of the brain followed the disintegration of some pneumatic cells located beyond the limits of the mastoid process, near to the occipital bone. Blau (l. c.) in 1,000 cases of intracranial complications cites only one case of brain abscess in which the pus infected the middle cranial fossa by disintegration of pneumatic cells in the same region. Usually, as we know, the pus enters the middle cranial fossa through the tegmen of antrum or tympanum, according to Blau, in 134 cases among 234 abscesses of the brain (l. c. page 118).

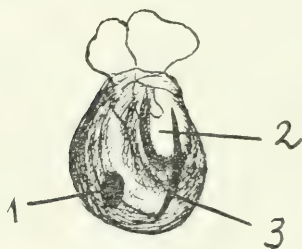


FIGURE 1.

1. Old perforation.
2. Bulging of the drum sack.
3. Incision.

LXXXIII.

SOME GENERAL EFFECTS OF LOCAL ANESTHETICS ADMINISTERED AS IN TONSILLECTOMY.

BY ELLISON L. ROSS, PH. D., M. D.,

CHICAGO.

Symptoms of the systemic effect of local anesthetics frequently appear during the removal of tonsils. Sometimes this effect is hardly noticeable and at others it is very pronounced. In the mildest cases there is a tendency to tremble and a feeling of weakness after the operation. In the more severe cases this is intensified, definite systemic effects are noticeable; faintness appears, the face turns pale and may show some cyanosis, breathing is rapid and the pulse is scarcely perceptible to the touch. The occasional severity of these symptoms of intoxication has aroused interest, and the author determined to ascertain as far as possible the primary cause and the mechanism of these reactions.

The general toxic effect of cocain and the occurrence of a high degree of susceptibility to the drug of some individuals are of common knowledge. This has led to the exercise of great care in its use. Today few inject cocain for any purpose. Its use is confined chiefly to surface applications in limited amounts. It is common practice to paint the pillars of the tonsils with a 10 to 20 per cent cocain solution before the local anesthetic is injected for a tonsillectomy. The local anesthetic usually injected is procain (novocain) with some adrenalin. So intoxication may arise from one, two or three drugs acting individually or synergistically.

The problem is emphasized by Dr. T. J. Harris¹ in the report of a fatality. Cocain, gr. 1/12, and adrenalin, 8 to 10 minims, were injected. Death occurred quickly. The heart seemed to stop a considerable time before the respiration. The autopsy showed enlarged glands in the axilla and groins and the thymus weighed 18 grams. Dr. Harris reported the cause of death as the result of "overdilated right auricle due

to an enlarged thymus gland, with cocain and adrenalin as an exciting cause." In the discussion Dr. Hubbard reported a death from 15 to 20 minims of adrenalin and thought that Dr. Harris' case resulted fatally from the adrenalin. Dr. Swain stated that he had treated a case of enlarged thymus with adrenalin and could see no "incompatibility between the two." He was inclined to think the cause of death of Dr. Harris' patient was the adrenalin. Dr. Roe, in discussing the case, thought that the death was due to hypersensitiveness to cocain. Dr. Harris, in closing, stated that he thought that the cocain had no deleterious effect in his case. This discussion by experienced men of the action of commonly used drugs which resulted in death emphasizes the incompleteness of our knowledge of the clinical action of these drugs and the importance of the subject.

Everyone recognizes the toxic effect of cocain and its variability in different individuals. Adrenalin, alone, is used rather freely with little thought of any toxic action. However, Buxton² in his book on anesthetics says that even minute doses of adrenalin are said to cause unpleasant effects—e. g., giddiness, fainting and collapse. Mortimer³ discussed the dangers from adrenalin as viewed by the pharmacologist and the harmlessness of the drug as viewed by the physician. He concludes that the question is an unsettled one, and the safest procedure is not to inject adrenalin but use it for surface applications only. Allen⁴ in his book on anesthesis states that adrenalin may prove a distinct advantage after cocain. He quotes J. M. Berry as agreeing with him. He mentions Thriss, Niles and Muhlberg and Petrow as considering the action of adrenalin to be that of a beneficial stimulant after cocain. As far as we are able to learn, no clinician has suggested synergy.

Pharmacologists agree that cocain and adrenalin act synergistically on blood pressure. If adrenalin and cocain are injected into a vein the rise in blood pressure is more than the sum of the increases produced by the adrenalin and cocain if injected separately and a considerable time apart. Cocain seems to sensitize the sympathetic nervous system so that the stimulation by adrenalin produces a much greater reaction. These facts led us to suspect considerable rise in blood pressure due to drug action during the process of a tonsillectomy under

local anesthesia. From the clinical side, there seems to be some doubt on this point. It is usually hard to feel the pulse when a toxic disturbance results from injections of a local anesthetic, and the common conclusion is that the blood pressure is low. Naturally, with fainting and respiratory disturbance, a low blood pressure is expected, since these are marked symptoms always present with severe hemorrhage. Usually with moderate increases in blood pressure there is an increase in buoyancy and a lack of any disturbance of respiration.

Recently the author made a series of experiments⁵ on dogs to determine the effect of the local anesthetic when administered in the usual clinical manner. In Test I a number of chlorotoned animals were injected in the tonsillar region with a dose of adrenalin. As soon as the effect of this drug wore away a dose of adrenalin with cocain was injected. Observations were made on the arterial and intracranial venous blood pressures, heart action and respiratory rate. The average results are represented on the chart. Test II was made on another group of dogs, in which moderate doses of morphin and atropin were given half an hour before the same treatment as the first group. The average results are represented parallel with those from Test I. Test III was made on a third group of dogs. This group was given the usual dose of adrenalin in the usual way, used in Test I, and after the effect of this had worn off cocain was injected. The results are represented parallel with those of the preceding tests. The values are expressed in per cents of the normal.

Tests I, II and III determine the effect on arterial and intracranial venous blood pressures, heart action and respiratory rate, of injections in the tonsillar region of adrenalin, cocain, adrenalin and cocain, and adrenalin and cocain preceded by morphin and atropin. The arterial pressure was increased 8 per cent by adrenalin alone, 83 per cent by cocain, 223 per cent by adrenalin and cocain, and 202 per cent by adrenalin and cocain preceded by morphin and atropin. From this the synergistic action of adrenalin and cocain will be calculated as 132 per cent. The injection of morphin and atropin affected the combined action of adrenalin and cocain relatively very little. It is striking that the arterial pressure is so enormously increased.

The intracranial venous pressure was unaffected by adrenalin, but increased 100 per cent by cocain, 467 per cent by cocain and adrenalin, and 282 per cent by adrenalin and cocain preceded by morphin and atropin.* This enormous increase in venous pressure due to adrenalin and cocain is more than twice as great as that taking place in the arterial system. This condition of affairs is very significant. Whenever the intracranial venous pressure increases twice as much as the arterial pressure, there is sure to be intracranial circulatory stasis and a resulting asphyxia of the central nervous system. Asphyxia of the respiratory center would increase respiration and give a feeling of smothering to the individual. Our tests show that the rate of respiration was increased 100 per cent by adrenalin and cocain. A feeling of smothering is a common complaint where there is any considerable reaction from the use of cocain. The failure of adrenalin and cocain to produce any increase in respiration when following morphin and atropin is due to the action of morphin to reduce the sensitiveness of the respiratory center. Asphyxia of the higher parts of the brain would lead first to a feeling of uneasiness and some degree of excitement, which would quickly be replaced by a feeling of faintness and dimness of vision. This is the common experience of the individual who suffers a reaction from the use of adrenalin and cocain. The changes in the heart action caused by these injections are of little importance. The slight slowing of the heart action with an increased amplitude of beat seems to be only compensatory changes.

The above tests were made by injecting adrenalin and cocain. On account of deaths resulting from the injection of cocain, it is chiefly used in the practice of medicine only on exposed surfaces. The question as to whether the local application of cocain in the usual manner about the tonsils would exert any influence on later injections was yet to be answered. Test IV was made on a fresh group of dogs. Adrenalin was injected into these dogs, and after its action had worn away, the throat was swabbed with 20 per cent cocain. Shortly after the cocain a second injection of adrenalin was made. Test VI was made on another group of animals, which was given adrenalin followed by swabbing with cocain. This test enabled us to measure the action of cocain alone. Tests IV and VI

furnish data by which we might measure the influence of throat swabbing with cocain on the action of injected adrenalin.

The arterial pressure was unaffected by the dose of adrenalin used on this group of dogs when no cocain had been applied. Swabbing with cocain after the action of the first dose of adrenalin had worn away produced an increase in arterial pressure of 16 per cent, and the repetition of the dose of adrenalin closely following the cocain produced an increase of 137 per cent. The synergistic effect of the cocain when swabbed on and the adrenalin injected was very great on the arterial pressure.

The changes in intracranial venous pressure produced in Tests IV and VI are similar to those of the arterial pressure. The first injection of adrenalin produced a very slight drop (7 per cent), and the second injection produced an increase of 193 per cent. The rise in intracranial venous pressure was very much greater than the rise in arterial pressure. The same condition is brought about by swabbing with cocain as by injecting with the cocain.

The question that naturally arises is whether swabbing of the throat with cocain, novocain and adrenalin would produce an important rise in blood pressure. Test V was made on a new group of dogs. These dogs were injected with adrenalin and novocain. After the action of these drugs had subsided cocain was swabbed on the throat, and following this the first injection was repeated. This test gave approximately the same results as without the use of novocain, namely, a rise in blood pressure.

The results of these six tests indicate clearly that cocain swabbed on the throat preceding an injection containing adrenalin has a very powerful influence on arterial and intracranial venous pressure. The intracranial venous pressure is much more increased than the arterial pressure. This we consider as the possible primary cause and mechanism of temporary disturbances after the use of these drugs.

The fact that all of the preceding tests were made on dogs and not men, and the fact that all of the dogs were under general anesthesia, make it possible that the conclusions reached from the described experiments are not applicable to the con-

ditions met with in the removal of tonsils under local anesthesia in the usual manner. To test this possible objection, blood pressure measurements were made on a number of patients during tonsillectomy.

Systolic blood pressure measurements were made at intervals during tonsillectomies, done by four different operators. The first measurement was made in the operating room before the operation. The second measurement was made just before the dissection of the first tonsil. The third measurement was taken just before the dissection of the second tonsil and the last measurement occurred at the end of the operation.

The drugs used by all operators were the same. Cocain and adrenalin were swabbed on the throat before the injection of novocain and adrenalin. The amount and method of administration of the drugs were different with the operators. Operator A swabbed the throat with 8 per cent cocain and a small amount of adrenalin. He injected $\frac{1}{2}$ per cent novocain containing about 5 per cent of adrenalin (1:1,000). He always injected both sides before starting the operation. All of his cases received $\frac{1}{8}$ grain of morphin and $\frac{1}{150}$ grain of atropin three-quarters of an hour before the operation. Operator B swabbed the throat with 10 per cent cocain with 50 per cent adrenalin (1:1,000) before injecting novocain—1 per cent, containing a little over 1 per cent adrenalin (1:1,000). One side was injected and operated on before the second was injected. Operators C and D swabbed with 20 per cent cocain and a small amount of adrenalin before 2 per cent novocain and about 4 per cent adrenalin (1:1,000) were injected. In some cases both sides were injected before any operating was done, and in others one side was injected and the tonsil removed before the other was injected. In Table I is recorded the individual initial, maximal and final blood pressures taken.

Referring to Table I, it is clear that there was always a rise of blood pressure. The amount of rise in blood pressure was quite variable, from 20 mm. of mercury to 114. In 18 cases out of the 23, the maximal pressure occurred before the removal of the second tonsil.

Tables II and III contain the average measurements. In Table II the results are expressed in millimeters of mercury, and in Table III they are expressed in percents of the normal.

The average blood pressure for all 23 cases were 119.8 mm. before operation, 160.8 mm. at the height of rise and 140.8 mm. at the close of the operation. The average maximum of rise was 34.2 per cent and the average increase in blood pressure at the end of the operation was 17.6 per cent. This maximum of increase is considerable and should be considered as a possible source of danger in some cases. Certainly, these measurements indicate that efforts to prevent mishap from the use of local anesthetics should not be directed toward increasing the blood pressure but toward preventing a rise in it.

There were three cases in this series that had rather severe fainting reactions. The average blood pressure of these before operation was 128 and the average pressure raised to 194 mm., and at the end of the operation the average blood pressure was 162 mm. of mercury. The maximum of pressure was an increase of 51.5 per cent, and the final pressure was 26.5 per cent above the initial pressure. These few cases certainly substantiate the view arrived at by animal experimentation that the rise in blood pressure is the cause of the temporary disturbances produced by local anesthetics.

Some operators consider that there will be less reaction from the local anesthetics in tonsillectomies if one side is injected and operated on before the other side is injected. Of the series of cases, 13 were so managed and 10 received injections on both sides before dissection was begun. The first group began with a blood pressure of 115.9 mm., which raised to a maximum of 156.8 mm. and ended with a pressure of 131.8 mm. The second group began with a pressure of 124.9 mm., which raised to 166 mm. and then finished with a pressure of 152.4 mm. The maximum of rise in the first group was 35.3 per cent, as compared with 32.9 per cent in the second group. The final pressure in the first group was 13.7 per cent above normal, while that of the second group was 22 per cent above normal. Such results do not argue very strongly in favor of injecting one side and operating on it before injecting the second side.

There were some variations in the work of the several operators. Operator A used more adrenalin and less novocain than the other three, and he always injected both sides at once. Operators C and D used more adrenalin and novocain than

Operator B. The maximum of increase for Operator A was 52.5 per cent, for Operators C and D 36.5 per cent, and for Operator B 26.5 per cent. These values grade in the same general ratio as the amount of adrenalin used. Other variations in the performance of the operators and changes of blood pressure did not seem to be of significance.

SUMMARY AND CONCLUSIONS.

The administration to animals of local anesthetics with the usual clinical technic practiced in the removal of tonsils, produces marked circulatory changes. The arterial pressure is greatly increased, as would naturally be expected from the well known synergistic action of cocain and adrenalin when injected into the blood stream. The intracranial venous pressure is relatively very much more increased than the arterial pressure. The arterial pressure was increased 223 per cent, while the venous pressure was increased 467 per cent. If either cocain or adrenalin be omitted the circulatory changes are negligible. In the previous paper⁵ it was pointed out that the great variations in blood pressure were capable of producing marked mental and respiratory disturbances.

Measurements of blood pressure in people undergoing tonsillectomies in which cocain, adrenalin and novocain were used indicate a regular rise or blood pressure of considerable amount but subject to wide individual variation. Those cases which experienced considerable reaction showed much greater increase in blood pressure.

These observations lead us to conclude that the minimum of cocain, to be of any value as an anesthetic, is sufficient to greatly sensitize the body to the action of adrenalin on blood pressure. Therefore cocain should be eliminated completely wherever possible. When cocain must be used to reduce reflex action of gagging, it may be applied some time after the injection of the adrenalin and novocain, when no synergistic action will result. The amount of adrenalin with the novocain should be reduced to a minimum. The strength of the novocain injected up to 2 per cent seems to be of small significance.

TABLE I.

INDIVIDUAL BLOOD PRESSURE VARIATION DURING TONSILLECTOMY USING LOCAL ANESTHESIA.

No. Patient	B. P. Before Op.	Amt. Maximum B. P.	B. P. Time After Op.	
1 C-D	137	200	Before 2nd	Faintness.
2 C-D	120	234	Before 2nd	
3 B	130	150	Before 2nd	
4 B	115	160	Before 2nd	
5 A	150	194	Before 2nd	Both sides injected before operation. Very faint.
6 C-D	114	154	Before 2nd	
7 C-D	122	158	Before 2nd	
8 A	190	238	After 2nd	
9 C-D	105	130	After 2nd	Both sides injected before operation. Morphine-atropin before operation.
10 C-D	115	135	Before 2nd	
11 A	120	165	Before 2nd	
12 C-D	116	172	Before 2nd	
13 C-D	100	120	Before 2nd	Both sides injected before operation. Morphine-atropin before operation.
14 C-D	90	125	Before 2nd	
15 C-D	130	160	Before 1st	
16 C-D	110	135	Before 2nd	
17 A	135	190	Before 2nd	Both tonsils injected before operation. Morphine-atropin before operation.
18 A	126	150	Before 2nd	
19 C-D	122	150	After Op.	
20 C-D	100	140	Before 1st	
21 A	118	170	Before 2nd	Both sides injected before operation. Morphine-atropin before operation.
22 C-D	90	128	Before 2nd	
23 C-D	100	140	Before 2nd	
Average	119.8	160.8		

TABLE II.

AVERAGE BLOOD PRESSURE VARIATION DURING TONSILLECTOMY USING LOCAL ANESTHESIA.

	B. P. Before Op.	Maximum of B. P. Amt.	Increase	B. P. After Op.	No of Cases
All Cases	119.8	160.8	41.0	140.8	23
Fainting Cases	128.0	194.0	66.0	162.0	3
One Side Injection.....	115.9	156.8	40.9	131.8	13
Both Sides Injection.....	124.9	166.0	41.1	152.4	10
Operator A	121.0	184.5	63.5	176.0	6
Operator B	122.5	155.0	32.5	138.5	2
Operators C and B.....	111.4	152.1	40.7	127.0	15

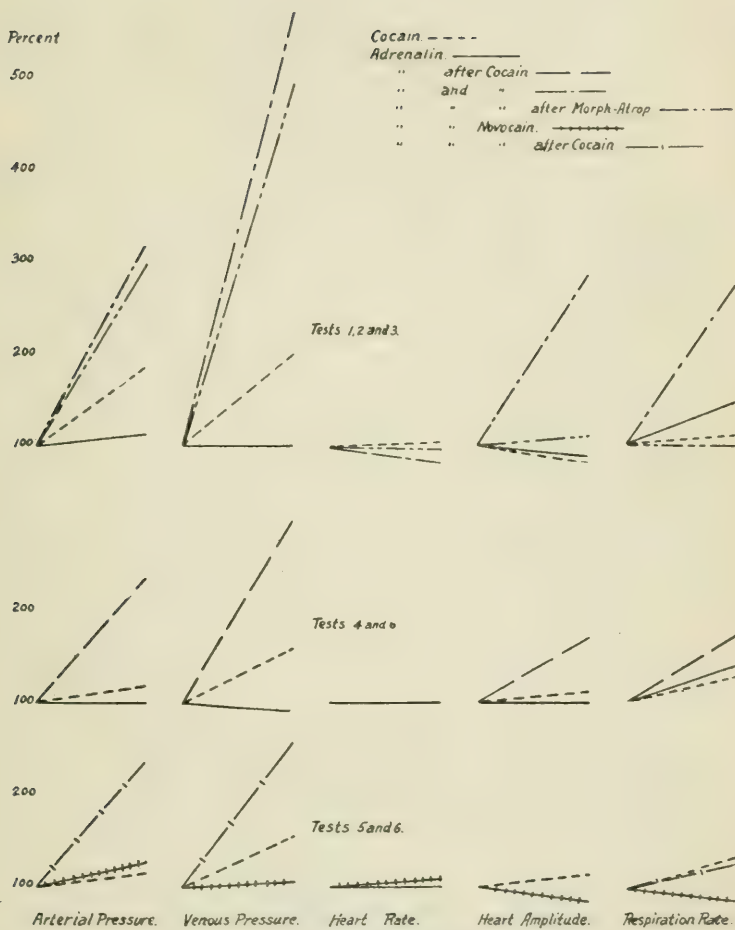
TABLE III.

AVERAGE BLOOD PRESSURE VARIATION DURING TONSILLECTOMY USING LOCAL ANESTHESIA.

Results expressed in percent of normal.					
	B. P. Before Op.	Maximum of B. P. Amt.	Increase	B. P. After Op.	No of Cases
All Cases	100	134.2	34.2	117.5	23
Fainting Cases	100	151.5	51.5	126.5	3
One Side Injection.....	100	135.3	35.3	113.7	13
Both Sides Injection.....	100	132.9	32.9	122.0	10
Operator A	100	152.5	52.5	145.4	6
Operator B	100	126.5	26.5	113.0	2
Operators C and D.....	100	136.5	36.5	114.0	15

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Animal Experiments.

LXXXIV.

TUMOR OF THE CAROTID GLAND WITH STOKES-ADAMS SYNDROME.

By G. W. BOOT, M. D.,

CHICAGO.

The members of this society will remember the patient I demonstrated before them in 1921 as a case of tumor of the neck with Stokes-Adams syndrome. He was a mulatto, aged 42, who was admitted to Cook County Hospital complaining of pain in the left side of the neck and cramps of the muscles on the same side of the neck. His symptoms had been present for the preceding six months. The onset was sudden, with cramps in the muscles of the neck and unconsciousness lasting about a minute. Breathing was very difficult during the attack and the neck was painful. Since that time he had from one to three attacks daily. At times he would have intervals of several days during which he was free from attacks. The pain in the neck and discomfort in breathing were relieved when the neck was flexed. At times the pain extended over the entire left side of the face.

He had acute suppurative otitis media in 1901, malaria in 1908 and gonorrhea in 1911. He had been married sixteen years and had a wife and two children, living and well. His hearing was impaired in the right ear. He vomited occasionally. He had lost 23 pounds during the past six months. He had sexual weakness to the extent that he had had no erections during the preceding six months. He had had no cough before the onset of these spells. He had had no pain in the chest and no night sweats.

Examination showed a small tumor about 2 cm. in diameter in the left side of the neck about its middle and anterior to the sternomastoid muscle. This tumor was painful, and when touched the patient coughed and became weak. During the examination the patient became unconscious for about 30 seconds and frothed at the mouth but had no general convulsion. There was paralysis of the left vocal cord.

Diagnosis by the intern, extrinsic tumor of the larynx with epilepsy.

Examination by the resident confirmed the above findings. His diagnosis was extrinsic tumor of the larynx, left abductor paralysis, with possible epilepsy or hysteria.

Examination by myself was difficult for the reason that any attempt to use the throat mirror brought on a convulsive action of the muscles of the neck and larynx with difficult respiration, and it was only after several attempts that I was able to see that there was no movement of the left vocal cord outward. During this examination I discovered that pressure on the tumor would bring on a convulsive attack during which his pulse became slow. On listening to his heart with a stethoscope, the sounds became faint, and if the pressure on the tumor was long enough continued the pulse could not be felt at the wrist, the heart sounds stopped and the patient had a general convulsion. The condition was that of Stokes-Adams syndrome from vagus inhibition.

The tumor was operated on under the impression that I was dealing with an enlarged lymphatic gland that was in intimate connection with the vagus so that there was partial paralysis of the left recurrent nerve from pressure.

The operation proved to be much more difficult than was anticipated. The tumor was found to lie in the bifurcation of the carotid and intimately to surround the common, internal and external carotids and internal jugular vein so that it could not be separated from them. It was necessary to ligate the common carotid, the internal and external carotid and the internal jugular vein before the tumor could be removed.

I do not know whether the vagus was divided or not, but it probably was severed. Trouble was anticipated from this source but apparently nothing of consequence occurred. His symptoms remained much the same after the removal of the tumor except that he had no further attacks of Stokes-Adams syndrome, and his pulse was more rapid. He coughed a good deal and died about a week after the operation.

At the postmortem it was found that he had acute edema of the epiglottis, glottis, vocal cords and larynx generally; large ulcerative, purulent tubercular cavity of the upper lobe of the right lung; multiple abscesses of the lower lobe of the

left lung; edema and hyperemia of both lungs; acute purulent tracheobronchitis; apical fibrous pleurisy on the left side; right hydrothorax; fibrous perisplenitis; cirrhosis of the liver and fibrosis of the spleen.

The tumor removed was about 3 cm. long, 2 cm. wide and 1.5 cm. thick. It could not be separated from the carotids. The microscopic diagnosis of the tumor removed was in doubt. The pathologists finally concluded to call it carcinoma of the carotid gland.

The carotid gland is a small body about the size of a navy bean, lying at the bifurcation of the common into the internal and external carotid arteries. Its beginning has been seen in the 4 cm. fetus. It is one of the chromaffin bodies, so-called, because of the affinity of its cells for the salts of chromium used in fixing. On this account sections of the carotid gland stain better when fixed in Mueller's fluid than when fixed in formalin. It is composed of cells with large protoplasmic bodies grouped into clusters or strands of various sizes, and lying in alveoli of connective tissue. Its cells somewhat resemble epithelium. It has many capillaries with thin walls. The carotid gland is connected to the carotid artery at its bifurcation by a sort of pedicle composed of fibrous and vascular structures. Its blood supply is by three or four small arteries that enter at its lower pole. A corresponding number of veins leave at its upper pole. Its nerves are numerous and come from several sources, among which are the vagus, the sympathetic, the hypoglossal and glossopharyngeal. Like other chromaffin organs, it possesses a few ganglion cells. The function of the carotid gland is unknown, but the paraganglions have to do with controlling blood pressure. The paraganglions include the tympanic gland, the carotid gland, Wiesel's cardiac paraganglion, the aortic paraganglion or Zuckerkandl's organ, and Luschka's coccygeal gland.

Tumors of the carotid gland are uncommon. The first tumor was reported by Marchand in 1891. Keen and Funke in 1906 collected 29 cases. Callison and Mackenty in 1914 brought this number up to 60. Reid in 1920 found reports of 66 cases and added 3 more from the clinic of Prof. Halsted of Johns Hopkins.

Tumors of the carotid gland may be benign or malignant. The benign tumors are simple hyperplasia, adenoma and angioma. The malignant tumors have been variously diagnosed as perithelioma, endothelioma, epithelioma, perithelial angiosarcoma, sarcoma, carcinoma, and so on.

These tumors vary in size from an almond to a goose egg. They are usually smooth and oval in outline, but sometimes are lobulated when they have been called potato tumors and are probably sarcomas or carcinomas. They are usually dark red in color on section but may be almost white or yellowish. Only one case is on record where they were bilateral. They occur in about equal frequency in the two sexes and on the two sides of the neck.

The symptoms caused by them are deformity; pressure symptoms, such as bruit and thrill, tinnitus aurium, hoarseness, cough and vocal cord paralysis from involvement of the recurrent nerve, contracted pupils from involvement of the sympathetic, dysphagia, and dyspnea.

In the diagnosis the following points are to be considered: The location at the bifurcation of the common carotid; movability laterally but not up and down; ovoid shape; usually smooth; usually single; transmitted pulsation; bruit and thrill; bulging of the lateral wall of the pharynx; pupils usually constricted; slow growth (cases have been reported lasting 8, 9, 16 and 40 years); absence of tenderness and pain, as a rule; slowing of the heart—i. e., Stokes-Adams syndrome; hoarseness and cough from vocal paralysis; pigmentation of the body in some cases.

Differential diagnosis should include consideration of the possibility of enlarged lymph glands—i. e., cervical adenitis, gland metastases in carcinoma, Hodgkins disease, branchial cyst, syphilitic enlargement of glands, gummata, tubercular glands, dermoids, aneurism. Many of these cases give the history of a lump in the neck for many years, with very slow growth. Finally the lump takes on rapid growth.

If these tumors are to be treated successfully they should be operated on early before they have become so intimately attached to the carotids that ligation of the carotids is necessary for their removal. The surgeon will rarely see them in this stage and will still more rarely make the correct diag-

nosis. Ligation of the common carotid is a serious operation though not necessarily fatal. I have ligated the common carotid twice, once for pulsating exophthalmus and once for tumor of the carotid gland. In the former case the patient recovered; in the latter, death was not the result of ligation but of the concomitant pulmonary condition.

In my case I feared the result of cutting the vagus, but my fears were not well founded. Before operating, my patient was given a large dose of atropin with the idea of suppressing the inhibition of the vagus. Possibly this was the reason why no serious trouble was encountered with this nerve.

The results in the cases operated on up to date have not been very creditable to the surgeon. The mortality is high, and laryngeal and other disturbances of a permanent nature should make one hesitate before operating. Among the serious complications that have resulted are hemiplegia, aphasia, paralysis of the facial nerve and of the hypoglossal nerve. These tumors recur rather frequently. Most cases have been operated on under mistaken diagnosis. In my case the operation was done in the attempt to relieve the patient of distressing epileptiform choking spells from which he suffered and which completely incapacitated him from work.

25 E. WASHINGTON ST.

KLEBS-LOEFFLER INFECTION OF THE MIDDLE
EAR AND MASTOID.

BY RUSSELL WEBBER, M. D.,

LARYNGOLOGIST AND AURAL SURGEON TO THE
WATERBURY HOSPITAL,
WATERBURY, CONN.

An examination of the literature shows but few recorded cases of true primary diphtheritic otitis. The textbooks mention cases of Klebs-Loeffler infection of the middle ear following cases of pharyngeal diphtheria, but practically no reference is made to Klebs-Loeffler infection not dependent on a known primary pharyngeal or nasal infection.

The first mention of the possibility of diphtheria of the middle ear unassociated with pharyngeal diphtheria is found in Billington's book published in 1889. Ballenger refers to cases of diphtheritic otitis sequelæ of pharyngeal diphtheria, but does not mention an infection primary in the ear. Phillips also dwells at some length on second diphtheritic infection and briefly dismisses primary infection in the ear by observing that it happens very rarely. Kerrison goes into the secondary otitis of diphtheria in some detail but does not refer to a primary infection.

In the usual form, an infection secondary to pharyngeal diphtheria, the otitis appears during the course of the disease. Epidemics of diphtheria vary greatly in the proportion of ear complications observed. Kerrison says that about 5 per cent of diphtheria cases develop some form of ear lesion. These lesions are much less extensive than those following measles and scarlet fever, and while there may be extensive destruction of the membrana tympani itself, mastoiditis is less apt to occur.

J. J. Thompson in 1914 reported eleven cases of diphtheritic or pseudodiphtheritic mastoid wound infection, all occurring during a period of six months. None of the cases had pharyngeal diphtheria. In but one of these cases did people coming

in contact with it develop diphtheria, guinea pigs injected with cultures from the wounds did not die, and no change occurred in any of the cases in which diphtheria antitoxin was used. Of all these cases, but two seem to be described as being positively Klebs-Loeffler infection. Thompson's finding that these cases were unaffected by the injection of antitoxin differs from the experience of other observers.

Pugnat describes primary diphtheritic otitis media and mastoiditis occurring as a sequel. All of the cases that he has observed cleared up rapidly with the injection of diphtheria antitoxin.

W. C. Bane of Denver, Colo., reports what he believes to be one of the few cases of true diphtheria of the ear. The course of the case was that of the usual acute otitis media. The patient had had pain in the right ear followed by rupture of the membrana tympani and drainage before being seen. Cultures taken from the aural discharge showed the presence of diphtheria bacilli and antitoxin was given. A general subsidence of the symptoms followed, but the bacilli persisted in the discharge for two months, the left ear finally becoming involved. This is one of the cases in which the injection of diphtheria antitoxin did not cause the disappearance of Klebs-Loeffler organisms from the discharge.

Blanchard reports a case of double diphtheritic otitis media. For eight days the case, apparently one of ordinary acute otitis media, did not respond to the usual treatment. He ran a high temperature and got progressively weaker. On the ninth day a peculiar exudate was noted in the ears, and when cultures were taken a Klebs-Loeffler infection was discovered. Antitoxin was administered and not only did the general symptoms subside very rapidly, but the discharge ceased in both ears within four days.

In Alfred Kahn's case there was a diphtheritic membrane surrounding and extending up and through the eustachian tube of the right ear. The process continued into the middle ear with destruction of the membrana tympani. An X-ray picture taken at the time showed extensive mastoid involvement, but operation was delayed until the effect of diphtheria antitoxin could be noted. Such marked improvement resulted

that operation was not resorted to, and the condition eventually cleared up completely.

Dr. Vinsonhaler of Little Rock, Ark., reports two cases of Klebs-Loeffler infection of the middle ear, one case eventually becoming diphtheritic mastoiditis. In neither case was there any pharyngeal diphtheria. In both cases all the severe general symptoms subsided with the injection of antitoxin, and within a few days diphtheria bacilli were absent from the discharge.

Two cases coming under my own observation are of interest.

Case 1.—C. M., aged 52. First seen February 5, 1920. Twenty years previous to this time patient had had an incomplete mastoid operation performed on the right side, and up till present illness had had no further trouble from this ear. About the middle of January, 1920, he had an attack of facial erysipelas, which had almost cleared up when seen. A few days before I was called to see him, his right ear began to discharge but he suffered no particular pain. On February 4th his temperature rose to 102, and he complained of severe discomfort in his right ear. When I examined the ear I found the canal filled with a thin watery discharge, but did not see any sign of anything resembling a diphtheritic membrane. There was no mastoid pain or tenderness. Examination of nose and throat was negative. I ordered frequent hot irrigations of boric solution and took a culture from the ear. When the laboratory report returned "Klebs-Loeffler," I felt sure a mistake had been made and my culture mixed with another, but a second culture gave the same result. Ten thousand units of antitoxin were given, and within thirty-six hours the patient's temperature dropped to normal and the discharge ceased. Two weeks later discharge recommenced. This time cultures were taken from the ear, nose and pharynx. All were positive for Klebs-Loeffler, though no membrane could be found in any of these sites. After a second dose of antitoxin the temperature returned to normal and the discharge diminished. A week later definite mastoiditis developed and the right mastoid was operated upon. At no time, then or thereafter, did the bacteriologist report presence of diphtheria bacilli, though frequent cultures were taken.

Case 2.—F. A., aged 3. Child was admitted to the Waterbury Hospital January 24, 1922, with discharge from both ears, pain and swelling in both mastoid regions, and a temperature of 104.4°. A double Schwartze was done by my colleague, Dr. Munger, immediately after patient was admitted. Both mastoids were very congested, granulation tissue was found in both antra, but there was no great outpouring of pus. Cultures taken from the mastoid antra were reported to be staphylococcus aureus. For days following operation there was no marked change in the child's condition. Temperature varied from 100 to 102 degrees. His movements were sluggish and his appearance was that of a very ill child. There were no symptoms to suggest intracranial complications. Eye grounds were clear and urine was practically normal. A blood culture was taken and staphylococcus aureus reported. Blood count was normal, aside from a reduction of the red blood cells, which were down to 3,000,000. It was decided to give the child an autogenous vaccine, made from the blood culture. The first dose was given on February 6th and there was a very marked reaction. Both mastoid wounds were again cultured and Klebs-Loeffler was reported in both but was absent from nose and throat. As the patient was already getting the vaccine for the blood infection and as he had had very severe reaction to it, but one dose of antitoxin was given and that but 5,000 units. Thereafter, the temperature rapidly subsided, although diphtheria bacilli persisted in the mastoid wounds until the 20th of March, when they were finally reported free. Child made an uneventful recovery.

The following conclusions may be drawn from a study of these cases:

1. That although the small number of recorded cases testifies to the rarity of the condition, diphtheria of the middle ear may occur without known previous Klebs-Loeffler infection elsewhere.

2. That a more careful bacteriologic examination of aural discharges in obscure cases may reveal the presence of diphtheritic infection otherwise overlooked.

3. That practically all cases of aural diphtheria respond in a marked manner to the injection of diphtheria antitoxin. In

not all cases does the Klebs-Loeffler organism disappear from the discharge, but in the cases reported the general symptoms did subside when antitoxin was administered. Hence in all cases where there is any question as to whether the infection is true Klebs-Loeffler or pseudodiphtheritic, antitoxin should be given.

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SOCIETY PROCEEDINGS.

THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, April 2, 1923.

THE PRESIDENT, DR. CHAS. H. LONG, IN THE CHAIR.

Nasal Obstruction Which Apparently Had Affected His Mental State.

DR. C. H. LONG presented a patient who had been operated on for nasal obstruction, which apparently had affected his mental state. The patient insisted that there was a cartilage which obstructed his left naris and that he was unable to get free passage of air through his nose.

Foreign Body in the Esophagus.

DR. JOHN A. CAVANAUGH reported a case of foreign body in the esophagus in which a diagnosis of carcinoma of the esophagus had been made, and presented skiagraphs showing the hourglass appearance and obstruction of the esophagus. The history was that the patient awoke one morning and had difficulty in swallowing with regurgitation of food. He had no knowledge of having swallowed a foreign body, but had been on a "spree" for a few days and missed a small artificial denture from the front of the mouth which he thought had been lost. Roentgen examination revealed the cause of the obstruction and the plate was removed without much difficulty. The patient made an uneventful recovery.

DISCUSSION.

DR. GEORGE W. BOOT thought it was unusual for a patient to swallow a plate without knowing it and believed it was criminal for dentists to make such small artificial removable dentures. They should be large enough so that they could not slip into the esophagus.

DR. EDWIN MCGINNIS recalled a case he had seen where the patient had swallowed a plate containing three teeth. The

patient developed hoarseness and later difficulty in breathing but had no knowledge of having swallowed a foreign body. Upon examination the physician saw the upper edge of the denture and removed it with forceps.

Paper: "Tumor of the Carotid Gland With Stokes-Adams Syndrome."*

By GEORGE W. BOOT, M. D.

DISCUSSION.

DR. OTTO STEIN thought these tumors were probably not as common as they might seem and that they were usually diagnosed late, frequently on the operating table.

He recalled a case which he had considered a tumor of the neck of indefinite origin. Upon attempting to operate they found a dense, vascular and adherent mass, and the disturbance caused by manipulation indicated that the tumor had something to do with the pneumogastric. The effects were so pronounced that operation had to be discontinued.

Dr. Stein said he would hesitate to operate in such a case, for the mortality is enormous and the possibility of a patient living for a long time with such a gland is more favorable than that of recovery following operation. Most cases are of malignant type when seen. The location of the mass, at the bifurcation of the external and internal carotid artery so closely associated with the nerves, the hypoglossal and superior cervical plexus lying on the anterior surface, makes dissection very difficult without producing some disturbance of the nerves. In the early stages this difficulty would not be so great, but the cases are seldom seen early, and the intimate association with the carotid made them practically hopeless operations. The ability to diagnose this condition and differentiate it from many other things was of importance, for many things lead to suspicion of the carotid gland. He was of the opinion that such growths should be left alone.

DR. JOSEPH C. BECK asked Dr. Boot to explain the connection between the epilepsy and the growth. He thought perhaps the relation of the sympathetic ganglion to the tumor was of some importance in this connection, along the line of the work of Jenesco regarding the ligation of the carotid to

See page 1241.

cure epilepsy. He believed he might be as lethargic as Dr. Stein in leaving such a growth alone.

He also asked Dr. Boot if he did not find in the literature that the carotid gland was related to the polyglandular system. In his opinion, radiotherapy might have been given a trial in this case.

DR. BOOT (closing) said that another physician saw the patient and used radiotherapy before he took charge of the case. He thought Dr. Stein's advice to leave such cases alone was very good. The time to remove these growths was before the tumor got large enough to involve the carotid, but that was before he sought the advice of the physician, as a rule.

He was glad to hear Dr. Beck mention the work of Jenesco but thought it had been entirely to the discredit of the profession and believed that Jenesco himself had changed his opinion.

Dr. Boot believed the cause of the epilepsy in his case was anemia of the brain from the compression of the growth, that the sympathetic ganglion had nothing to do with it, but that it was a Stokes-Adams syndrome. They could neither feel the pulse nor hear the heart sounds with the stethoscope before the convulsion.

Paper: "Congenital Atresia of the Posterior Nares."*

By JAMES E. LEBENSOHN, M. D.

DISCUSSION.

DR. ROBERT SONNENSCHN said he had nothing to offer in the way of criticism or in addition to the subject matter, but congratulated Dr. Lebensohn on his clear and logical presentation of the subject and the society on the acquisition of so valuable a member.

The point regarding the lack of dependence of ear conditions upon obstruction of the nares was also emphasized at the last meeting of the Illinois State Medical Society by Dr. Shambaugh. Dr. Sonnenschein thought it important to remember that while obstructions of the nose may influence middle ear conditions when they favor extension of the infection by way of the eustachian tube, it is not necessary to

See page 1128.

remove every little spur when operating, on the assumption that it will produce difficulty with the ear. He had recently seen a case of marked deviation of the septum to one side, but this patient had never complained of obstruction, as frequently happens. In this instance the patient asked whether the septum should be operated upon and said she had been told by another man that she must have an operation because she might get a sinus or ear infection some time in the future. As there was no obstruction Dr. Sonnenschein advised against operation.

DR. JOSEPH C. BECK said he did not know this condition was so rare and could add six cases that he had operated on in children. He offered a suggestion regarding the reclosure which the patient complained of. The electric burr is not very easily managed in the nares, even with the finger as a guide, but he believed the perforation could be very easily made by means of a large rasp which would penetrate most of the bony obstruction. If not, a hand chisel could be used for the first opening and then a rasp could be used to make the hole as large as necessary. Having completed this, a hard rubber plug, which is flattened so that it will not obstruct the nasal spaces, is drawn by means of a catheter into the nose and fastened in place by means of tape drawn into the opposite nostril with a bit of gauze over the columella. He had removed a bilateral obstruction that had existed since childhood in this manner, with good results and great improvement in speech, but ten minutes after the patient was returned to his room he was called to see him because the man was in a fit of anger because he could not talk to suit him. The nasal twang distressed him, and he insisted upon stuffing cotton into his nose so that he could get his old manner of speech back.

DR. JOHN CAVANAUGH recalled the case of a child of about twelve years in whom the parents would not permit operation. The tonsils and adenoids had been removed, the latter on three different occasions, the inferior and middle turbinates had been removed and the mother told him the doctor had said something about removing some of the ethmoids on that side. Dr. Cavanaugh discovered the postnasal obstruction in trying a through and through irrigation, but the mother would not consent to further operation.

DR. FRANK J. NOVAK, JR., asked whether there was a true anosmia, and whether Dr. Lebensohn had introduced any odoriferous substance directly into the nasal cavity. He thought that only in this way could one tell definitely whether or not there was a true anosmia.

DR. JAMES E. LEBENSOHN (closing) thought the point Dr. Sonnenschein emphasized in regard to the ear conditions being only secondarily due to nasal obstruction was important.

He was very glad Dr. Beck mentioned having such a large number of cases and felt that many cases were not reported because their rarity was not realized. He thought the profession should be more alive to this condition and thus avoid doing secondary adenoidectomies and other unindicated operations.

DR. ELLISON L. ROSS read his inaugural thesis on

Paper: "Some General Effects of Local Anesthetics Administered as in Tonsilectomies."*

By ELLISON L. ROSS, M. D.

DISCUSSION.

DR. OTTO STEIN asked whether there was an attempt at controls in the experiments and whether the position of the patient was taken into consideration, whether they were upright or reclining. He believed the injection of water or salt solution would produce an effect on the blood pressure.

DR. J. GORDON WILSON said that the knowledge of the synergistic action of drugs had been known for a long time. It had, for example, been recognized that two drugs together may act on a structure—e. g., the intestines with a much greater potency than the summation of the actions of each separately. The explanation is not known. But the synergistic action of adrenalin with cocain is not to be grouped with the similarity of actions of so-called adjuvants. That cocain and adrenalin together act synergistically appears to have attracted little attention from clinicians. The great importance of the consideration of such action was shown by Dr. Ross' paper and might well make one thoughtful.

See page 1229.

The current opinion of the conjoined action of adrenalin and cocain has been recently expressed in a textbook by a distinguished clinician, namely, that (1) by injecting adrenalin one contracts the blood vessels and stops or lessens absorption of cocain, (2) that adrenalin acts as a physiologic antidote to cocain. To the suggestion that adrenalin acts as a physiologic antidote to cocain, Dr. Ross' experiments are in contradiction. The very great rise in arterial and intracranial venous pressure from the combined action of cocain and adrenalin makes one pause and consider how little we have realized how great a disturbance of cranial circulation we have been bringing about with these so frequently and freely used drugs. It makes one pause and consider whether the advantages of swabbing a throat previous to tonsil operations in which adrenalin and novocain are injected are not offset by the disadvantages of this synergia.

Further, these experiments indicate the explanation of the symptoms frequently seen in operations following the conjoined application of cocain and adrenalin, and that we are not dealing with a diminished arterial pressure but a rise of arterial and venous pressures.

DR. EDWIN MCGINNIS thought the paper was very illuminating and interesting. It bore out some clinical findings he had observed in using local anesthesia in nasal and tonsillar operations. Some years ago he eliminated the use of adrenalin in doing tonsillectomies under local anesthesia. He did not realize the synergistic action of the drugs, but used them with a definite idea in view. The reason for using the adrenalin was to reduce the absorption of the cocain and to reduce the bleeding at the time of operation. He decided that in doing operations under local anesthesia he did not care how much bleeding there was and that the adrenalin could be eliminated. He noticed that with the topical application of adrenalin in the nose he got as much upset at times as with cocain alone, and he now paints the mucous membrane once with 10 per cent cocain and then injects the novocain. In this way he gets less upset for the patient, probably because there is less synergistic action from the novocain with the elimination of the adrenalin.

DR. HARRY L. POLLOCK said he would like to see Dr. Ross experiment with the cocain application. Formerly they used the 5 to 20 per cent cocain in operating on the nose, but had a great many of the toxic effects, particularly with the weaker solution, the 5 and 10 per cent, which they attributed to the fact that they got more absorption. The higher the percentage of cocain the greater astringent action on the blood vessels and the less the absorption. For several years they had used nothing but the cocain flakes, dipped in very weak adrenalin solution and applied to the mucous membrane. Since they have followed this plan the toxic effect has been much less pronounced. In doing a suspension or in nasal work they use the cocain flakes in large amounts and have had very few untoward effects as compared with the cocain solution. He believed this was due to the astringent action on the mucous membrane and the small blood vessels preventing absorption into the system.

DR. ROBERT SONNENSCHNIG thought this was one of the most valuable papers that had been offered before the society in a number of years. Many of the statements made corroborated things which, from a clinical standpoint, he had felt for a long time. Warned by an experience in the Vienna Clinic some eighteen years ago, where two patients died immediately after a submucous injection of cocain, he had never used it. In recent years he had used no cocain at all except in those patients who were so sensitive that the introduction of the needles was very painful, or where the pharyngeal reflex was so active that it was necessary to use it. Preliminary to nose and throat operations he had seldom used morphin, because he could not see that those patients who had received morphin had any less difficulty than occurred in those cases in which it was entirely omitted but that the opiate often did cause nausea or vomiting. He thought that the simpler the technic and the less drugs that were used, the better for all concerned. The substitution of apothecin made cocain practically unnecessary for throat work. They had found very few indications of syncope or other untoward symptoms during tonsillectomy since using a modified technic with alcohol, regarding which a paper would soon be read.

DR. GEORGE W. BOOT spoke of a tonsillectomy where the operator brushed over with a 5 per cent cocain and then injected a 1 per cent novocain to which adrenalin had been added. The patient had a blood pressure of about 280 at the time of operation, but the operation was performed because pus was exuding from every crypt. During the operation the patient developed great anxiety and complained of intense pain over the region of the heart. She went into collapse, and they thought she was dead, but after giving a hypodermic of morphin the distressing symptoms passed over and the next morning she was all right. She died a year later of apoplexy.

DR. FRANK J. NOVAK, JR., said that in the Army when they were unable to obtain novocain they used apothecin with good results. They had unpleasant results following the use of cocain, so they used apothecin topically for all nasal operations. While the induction of anesthesia required a longer time, the anesthesia was as complete as that obtained with cocain. Since that time he had used 5 per cent apothecin topically with excellent results in all nasal operations. He has not used cocain since 1917.

DR. ELLISON L. ROSS (closing) said that their cases were in a partial reclining position when operated upon.

The psychic influence suggested as a cause for increased blood pressure was always to be taken into account. The injection of water in the place of local anesthetics had not been tried. Since a great part of the work described was done on anesthetized animals it was certain that the drugs have a very large proportion of the influence exerted.

He had not tried cocain flakes, but thought that the concentration would make no difference so long as adrenalin was injected afterward. The astringent action of the flakes of cocain would certainly tend to decrease the rate of absorption.

Dr. Ross' idea was not to condemn the use of adrenalin but of cocain. He does not use cocain at all in working on tonsils, but thought that if one wished it could be used after the injection of adrenalin and novocain. After three to five minutes the adrenalin would all be gone and so could not exert any synergistic action with cocain.

MEETING OF THE SECTION OF OTOLARYNGOLOGY OF THE COLLEGE OF PHYSICIANS.

PHILADELPHIA, PA., DECEMBER 20, 1922.

Extremely Large Nasal Polyp.

DR. GEORGE B. WOOD, the chairman, presented a specimen of an extremely large nasal polyp removed from a mentally defective boy of 19 years. The symptoms began during the last summer. The soft palate was pushed out to the level of the back teeth, and the polyp could be seen below the margin of the soft palate. It was removed by the following method: A rubber catheter passed along the floor of the nose was brought out of the mouth and a silk thread tied to the end of it. The thread was then brought back through the nose and tied to the middle of a wire loop. This wire was then pulled through the nose into the mouth and worked with the finger in the throat around the growth. The ends of the loop were passed through a Watson tonsil snare and the growth cut off close to its insertion under the middle turbinate, the polyp being withdrawn through the mouth.

Presentation of Two Patients With Cancer of the Nose and Throat. Treated With Radium.

BY RALPH BUTLER, M. D.

First case, J. E., widow, age 58. Was admitted to the hospital November 1, 1921, with a history of profuse watery discharge from the left nostril, which followed an attack of influenza in December, 1917. The discharge became mucopurulent and the nose was gradually blocked. No pain, no spontaneous bleeding, no loss of weight and no evidence of metastasis. Examination showed a tumor the size of a shelled walnut in the left nostril protruding into the nasopharynx and bleeds easily. Diagnosis by biopsy, papilliferous adenocarcinoma. Treatment, 25 mg. of radium in capsule was applied to the nasopharynx and 15 mg. to the left nostril for eight hours. Three weeks later the second application of radium was applied, 35 mg. to the nasopharynx and 15 mg. to the left nostril. Twenty-four days later breathing seemed a little

easier and the same dose of radium was used. This was repeated twenty-seven days later and then no treatment for two months. On March 23, 1922, 50 mg. of radium was applied for twelve hours. April 11, 1922, 35 mg. was applied into the left nasal cavity and was repeated one month later for eight hours. This time the mass in the nose seemed to be decreasing in size and the patient felt better. June 1, 1922, the patient complained of a purulent discharge from the right nostril since the last treatment. Radium was again applied to the nose June 22, 1922, 50 mg. for eight hours. Condition improved.

Second case.—S. S., male, age 54. Was admitted to the hospital November 26, 1919, with the following history: Two years previous patient noticed a deafness in the left ear, which continued to progress. No pain. On examination, an ulcerated area was seen in the nasopharynx. Physical examination negative. No metastasis, no loss of weight, blood Wassermann negative. December 11, 1919, 35 mg. of radium, in capsule, was applied for twelve hours. Six weeks later same treatment was repeated. No unusual effects except dryness of mouth and throat. March 18th, examination showed improvement, decrease in size of the growth and gain in weight. Radium was again applied September 15, 1921. Fifteen mg. applied twelve hours, March 21, 1922. April 11, 1922, reduction in the size of the growth but there was evidence of radium burn. April 27, 1922, patient had evidence of radium burn in the nasopharynx and a perforation of the soft palate. Radium treatment was applied September 14, 1922.

DISCUSSION.

DR. GEORGE B. WOOD stated that he had seen one case of squamous cell epithelioma, about the size of a half dollar, located on the posterior pharyngeal wall, completely disappear after one week's treatment with radium. He had seen the patient today, two weeks after the treatment, and only a little whiteness of the mucous membrane, resembling a leukoplakia, could be seen.

DR. GEORGE M. COATES stated that he saw a case of sarcoma involving the soft palate and lateral pharyngeal wall in 1916.

It was incised and radium inserted into the opening, with the result that the growth disappeared, just as in Dr. Wood's case, within a week. No return two months later. After a two weeks' business trip, the patient returned with the growth just as it was in the beginning, and it went on to the termination.

DR. FIELDING O. LEWIS spoke in favor of the Crosby Green operation when the malignancy involves the ethmoid or maxillary sinuses or both. It makes a very desirable means of applying radium to these regions. This method has been used in the radiologic department of the Philadelphia General Hospital. After the surgical procedure, bare tubes are introduced into the involved structure, and radium plaques are applied outside. The results have been most gratifying.

**Report of a Case of Brain Abscess of Otitic Origin With
Exhibition of Brain.**

BY GEORGE M. COATES, M. D.

The patient was a middle aged man, with a history of a running ear on the left side since childhood, but had not had any other symptoms until now. When first seen he had been ill for two weeks. Examination showed the external auditory canal was swollen closed, no particular signs of mastoiditis. He complained of vertigo and deafness. Operation revealed a noncellular type of mastoid, with cholesteatoma in the mastoid antrum and a small cell adjoining. The radical operation was without incident, and the patient was discharged from the hospital in ten days in apparently good condition. Fourteen days following the operation he developed a facial paralysis, no vertigo. Barany tests showed no response in the vestibular apparatus on the affected side. Secondary operation five weeks later showed that the labyrinth was necrosed, and the anterior wall was removed in one large sequestra. The whole petrous portion of the temporal bone was found destroyed. The patient had complained of severe headaches on the left side before the last operation. Brain abscess was suspected, but the marked destruction of the temporal bone was thought sufficient to account for the headaches. Paralysis of the tongue and pharynx developed on the tenth day following the

operation, and death occurred a few days later. On necropsy there was a diffuse, purulent meningitis covering the entire base of the brain. The petrous portion of the temporal bone was destroyed. The pyramid looked normal, but it was found so thin that a knife would puncture it at any point, going immediately into foul smelling bone. On a cross section of the brain, three abscess cavities were found in the temporo-sphenoidal lobe, one near the cortex. The whole left side of the brain was softened and flattened out.

DISCUSSION.

DR. GEORGE B. WOOD. A man in the army was shot in the ear about a year before he came under my observation. He was admitted to the Pennsylvania Hospital with evidence of intracranial pressure. The bullet was found to be lying in front of the petrous portion of the temporal bone on the level with the posterior internal wall of the tympanic cavity. The patient developed a series of brain abscesses, and later a hernia of the brain as large as a hen's egg, death following three months later. His whole frontal lobe was gone; there was scarcely any brain substance in it. The parietal lobe was in the same condition, as was also the occipital lobe. Practically one-half of the man's brain was gone, and yet he lived for three months. He was able to eat and attend to normal functions of life until a few days before he died.

DR. WALTER ROBERTS stated that the cases he had seen were similar to the one that Dr. Coates reported, but usually they had been tubercular in origin. He mentioned a conversation with Dr. Lewis, wherein there was a brain abscess in the frontal lobe secondary to frontal sinus disease, in which there were a few symptoms showing that a great destruction of brain substance can occur without symptoms.

DR. FIELDING O. LEWIS: The case mentioned by Dr. Roberts was a young man, who had a history of long continued purulent nasal discharge due to frontal sinus disease. Radical operation was performed. Convalescence was uneventful until three weeks following the operation, when he developed signs of pus in the frontal regions, swelling, redness and fluctuating of the skin, later a secondary operation. After exposing the

whole region of the frontal area, a small necrotic portion was found in the posterior plate of the left frontal sinus. On curetting this area, as much as two ounces of pus were evacuated from the brain. The patient had not had any eye or intracranial symptoms. He at this time is in splendid condition. No rise in temperature, mentally clear and feels good. The cultures from the pus showed streptococci and staphylococci.

DR. S. MACCUEEN SMITH stated that one of the most interesting points about intracranial abscesses, otitic in origin, involving the silent area, was the absence of symptoms. In his experience, about one-half of the brain abscess cases were discovered during the radical operation for the cure of chronic suppurative middle ear disease or mastoid disease. Before the advent of the epidemic of influenza his brain abscess cases were chiefly the result of the chronic suppuration form of middle ear disease. Since the influenza epidemic the largest number of brain abscess cases have been of the acute type. When there are symptoms of brain abscess and it is not possible to determine its location, his experience had been that inasmuch as the vast majority of all brain abscesses of otitic origin occur in the temporosphenoidal lobe one is justified in exploring the temporosphenoidal lobe before seeking for the abscess in any other locality. On operating for brain abscess he invariably follows the route of infection. He has never had a case live following an evacuation of pus from any part of the brain unless this method was followed. He was interested to see in Dr. Coates' specimen that there were several distinct abscess cavities. He does not share the German view that there is always a communication between the various cavities even though they may not be found.

DR. FETTEROLF: Several years ago Professor Behrens of New York reported a case of brain abscess and rather emphasized that you so often have them without symptoms. It was brought out in the discussion, and rather stressed by several men, that a careful study and search about the disposition of the individual would often show that some changes, in connection with other things, would tend to point out the fact that a brain abscess might be present. There is often a change in the psychology of the patient that might suggest a brain abscess.

DR. RALPH BUTLER wished to emphasize the point made by Dr. Fetterolf by citing a case which he had some years ago at the Lankenau Hospital. A man had been shot through the upper jaw and the ball was removed through the orbit. Nothing abnormal was suspected in the brain. He became very objectional in the ward, caused the nurses a great deal of trouble and his whole disposition was changed. An abscess of the brain was later found.

DR. S. MACCUEEN SMITH brought up the question of aphasia, especially what might be called optic aphasia, and related a case at the Jefferson Hospital, on whom he had operated for brain abscess. The patient had a particular type of aphasia. When he was shown a key he could not tell that it was a key. He would point to the door and show that it was meant to open the door. When shown a knife, would show that a knife was meant to cut something. He was of the opinion that optical aphasia is as important a symptom as a changed disposition.

DR. COATES, in closing, stated that his patient had shown both a changed disposition and aphasia. He thought that the abscesses shown in the brain were of an acute type because they were totally without capsule. The final diagnosis was a temporosphenoidal abscess, but at that time, in view of the extensive destruction of the petrous pyramid, and on account of his very poor condition, he did not deem it advisable to search for the abscess. His experience corresponds with that of Dr. Smith in that they should be opened through the line of infection.

Paper: "Congenital Atresia of Nostril."

BY WARREN B. DAVIS, M. D.

Female patient, born December 20, 1920, full term. Family history negative, excepting that the patient is one of twins. Normal in all respects, excepting atresia of right nostril. There was a shallow dimple where the right anterior naris should have been, only partial development of the right alar cartilage and more depression of the bridge of the nose than could be considered normal. Most probable cause of the malformation

was some continuous pressure on the lateral nasal area during early fetal life by contact with the other twin.

When the patient was five months old double "Y" incisions were made in the slight depression where the nostril should have been located. The skin flaps thus formed were separated from the underlying tissue. Incisions were made through the soft tissue, keeping the scalpel parallel with the nasal septum as it was carried posteriorly. The atresia was found to extend to approximately a coronal plane at the junction of the anterior and middle thirds of the lower turbinate. The opening was enlarged by the use of a nasal dilator. The skin flaps were placed over the anterior margins of the nostril and a small perforated hard rubber splint placed in the nostril and held in position by a silkworm gut suture passed through the floor of the naris and tied underneath the upper lip. Previous to operation epiphora had been continuous. There was marked improvement within three days after operation. Contraction of scar tissue after operation was difficult to control in spite of persistent treatment by special dilators. Secondary operation will be required in later childhood. It is believed that the ventilation secured by the formation of the small nostril will allow proper development of the nasal accessory sinuses and proper function of the nasolacrimal duct.

DISCUSSION.

DR. BENJ. D. PARISH stated that he had seen, about a year ago, an interesting case of congenital atresia of one nostril in a child two weeks old. The right nostril was entirely absent with the exception of a little tab of skin, which was found patulous coming from the inner canthus of the right eye. On probing, it was found to be apparently the lacrimal duct opening directly into this tube. The question arose whether there was any posterior opening under the posterior superior space and what might be done. The parents were quite willing and anxious to improve the cosmetic appearance, so the lacrimal duct was split and an incision made along the bridge of the nose and another incision along the cheek in order to use this flap for the right nostril. The result looked well following the operation, but as it was not his own case he was unable to tell what the final result was.

**A Radical Operation, Three Years Ago, for Adenocarcinoma of the
Ethmoid, Antrum and Middle Turbinate—No Recurrence.
(Presentation of Patient.)**

BY DAVID HUSIK, M. D.

(By invitation.)

In reviewing the literature of carcinoma of the inner nose and accessory sinuses he found that the authoritative opinion was that this condition is hopeless. Bloodgood has stated that not one of eighteen cases with malignant disease of the nares operated upon at the Johns Hopkins Hospital recovered and that there is no authentic case on record where recurrence has not taken place before the patient has left the hospital. Intra-nasal carcinoma exists in several forms. The order of the frequency is, 1, squamous celled; 2, cylindrical celled; 3, medullary celled, and, 4, adenocarcinoma. Alexander reporting on adenocarcinoma of the nares found one case aged 22 years, one aged 28 years, and one aged 33 years. The author's case is 32 years old. In 200 reported cases of intranasal carcinoma only 21 were undoubted cases of adenocarcinoma.

Jonathan Wright states that he has never seen in the nose and throat, where ablations of various growths are often made at repeated and long extended intervals, any evidence of a histologic change in type from the benign to the malignant in either papillomatous or adenomatous growths. This view is not held by others, who believe that adenomata and papillomata frequently become carcinomatous, and they account for the rarity of adenocarcinoma as being due to the rarity of intranasal adenomata.

The author reports the following case for the reason that the patient is free from recurrence three years after operation for removal of an adenocarcinoma involving the middle turbinate, antrum and ethmoid on the left side: A. W., age 32, reported to the University of Pennsylvania Hospital in May, 1919, complaining of left sided nasal obstruction and discharge. Examination showed the left nostril filled with a large grayish irregular polypoid mass, covered with a mucopurulent discharge, boggy to touch, and evidently attached to the middle turbinate. Transillumination showed a dark area below the

left eye and over two-thirds of the internal aspect of the antrum. Under cocain adrenalin solution enough of the growth was removed to establish nasal breathing. The patient disappeared but returned to the hospital November 2, 1919, with a severe left sided nasal hemorrhage. Examination at this time showed the left cheek and eye more prominent, and he had developed left sided deafness. The following biopsy study by Dr. Allan J. Smith was given: "The general appearance of the histologic structure would tend to its classification as an infiltrative adenocarcinoma of origin in a mucous secreting gland, such as occur in the mucous membrane of the nose." X-ray report showed disease of the antrum, anterior and posterior ethmoids. Fifty mg. radium in capsule was directly applied to the growth every seven days, and X-ray treatment was given by Dr. Pancoast with little effect. On December 18, 1919, radical operation for removal of the upper jaw was performed and the ethmoids were curetted.

Several days following the operation an acute otitis media developed which promptly cleared up. In March, 1920, there was seen evidence of recurrence in the region of the superior turbinate, which disappeared after applying 50 mg. of radium. The biopsy report was adenocarcinoma.

DISCUSSION.

DR. FIELDING O. LEWIS stated that Dr. Husik should be congratulated upon the splendid result, and he thought that the use of radium and X-ray was very timely used.

DR. GEORGE M. COATES: Dr. Husik, I think, quoted Dr. Bloodgood to the effect that these cases never recover. The Boston physicians have shown that they do recover. Their plan is the open face method, keeping the antrum open until the possibility and probability of recurrence is over.

Laryngeal Gumma—Ten Year Old Child.

BY JOSEPH D. SEIBERLING, M. D.

(By invitation.)

Evasive symptoms, the etiology of which is obscure, should direct one's attention to the probable factor being syphilis.

This is true especially just preceding or during adolescence. I could do no better than to refer to Hunter's Commentary on Syphilis in which he states, concerning laws of evolution of hereditary syphilis, "It is certain that an infant may pass through the secondary stage of inherited syphilis without ever presenting any symptoms. Those who have shown no symptoms in infancy may yet suffer in later life. It is clearly to be understood, however, that where this happens to adolescents, they suffer from a class of symptoms wholly different from those of infancy. It is not that the secondary stage has been delayed, but simply that it has passed through without ostensible disturbance. After a period of latency there will in many cases come a group of very peculiar affections, involving principally the sense organs."

D. S., 10 years of age, Russian parentage, born in the United States. Good health since birth. One uncle died of tuberculosis and another of cancer. Father 48, living and well. Served in the Russian Army. Two years ago had swelling of the thumb, which disappeared under mixed treatment. No Wassermann blood test at that time. Denies any knowledge of visible lesion. Has had occasional headaches, also fullness in the head. Blood Wassermann plus two. Mother 47, living. Had 13 pregnancies. The fifth, sixth and eleventh terminated at ten weeks, three and one-half and six months, respectively. First child died of hemorrhage. Third child died three weeks after birth from infection following circumcision. Eighth child died of scarlet fever at three years of age. Ninth child died of diphtheria at one year. Six children living and well. The subject of this report is the twelfth child. History of present illness: Six months ago, after returning home from school, noticed hoarseness, no sore throat. The hoarseness progressed until the voice became only a whisper. Was advised by family physician to have tonsillectomy. Reported to the Polyclinic Hospital in September, 1922. General physical examination negative. X-ray examination showed no metastasis in the lungs. Blood count shows: Erythrocytes, 4,610,000; leukocytes, 9,300; color index, 90. Blood Wassermann positive, plus four. Examination of the larynx showed left ventricular band and cord

infiltrated with neoplasm, which projected beyond the median line, somewhat irregular but smooth in contour, semi-nodular slightly and apparently fixing the cricoarytenoid joint. Right vocal cord slightly congested but otherwise normal and moves freely. Glottic chink narrowed to about one-third normal. Slight retraction of suprasternal notch on inspiration. September 21st, received 0.45 arsphenamin intravenously and this treatment has been continued once weekly up to the present time. Retrogression of the mass has been rapid.

DISCUSSION.

DR. GEORGE B. WOOD: This is an extremely interesting case, gumma in children being very rare, and especially gumma of the larynx.

DR. WALTER ROBERTS stated that in his experience the blood Wassermann reaction is often useless as a diagnostic measure. He has used potassium iodid as a therapeutic test in the diagnosis and was of the opinion that good results are often obtained when little anticipated by the use of ascending doses of potassium iodid, although repeated Wassermans have been negative.

DR. CURTIS C. EVES stated that Dr. Roberts' remarks called to mind a case he had treated some time ago. A man 70 years old with neoplasm of the larynx. Three negative blood Wassermans. No specimen had been removed for diagnosis. Patient was treated with radium and X-ray with little improvement. Later the patient's family physician administered potassium iodid for some asthmatic symptoms and the growth began at once to disappear.

DR. SIEBERLING: The patient's Wassermann is still plus four.

Paper: "A Case of Cerebrospinal Rhinorrhea."

BY JOHN EDWARD LOFTUS, M. D.

(By invitation.)

The author, after giving a complete review of all published cases and literature of cerebrospinal rhinorrhea, the compre-

hensive and thorough data as to the etiology, pathology, symptoms and diagnosis, reports the following case: A woman aged 40, was first seen January 5, 1922, after being treated three months for sinusitis. She was complaining of a constant dripping of clear fluid from the left nostril for the past three months. The patient has always been very stout. Present weight, 240 pounds. No history of any head injury. Present condition began October 23, 1921, after a very hearty laughing attack, which was followed by severe sneezing, patient becoming aware of a clear limpid fluid from the left nostril associated with a frontal headache extending to the vertex. The headache has been constantly present but diminishes somewhat when the flow increases. One-half an ounce of fluid escaped in one hour. The fluid was clear, odorless, tasteless and without sediment, alkaline reaction. The flow of the fluid is greatly altered or increased by the position of the head. When the head is dropped forward, the flow increases; when the head is in the upright position, the flow is less; when lying in bed with the head hanging over the edge, face downward, there is a drop of fluid every two seconds. During sleeping hours the flow is the same as during the day. The constant swallowing of the fluid at times has caused severe diarrhea. On three occasions the patient states that the fluid escaped from her left ear, that it came out in gushes and lasted a short time. This was not seen by the author. The fluid is also increased when the patient works, climbs steps, does any severe exercise or when she becomes angered and excited. Examination of her nose revealed a sharply deviated septum to the right. There is a spur at the base of the septum on the same side which touches the inferior turbinate. Tonsils are of average size and apparently normal. The sinuses were negative. The author was unable to determine definitely where the fluid made its exit but was of the opinion that it came from the posterior portion of the roof of the nose and not from the middle meatus. Examination of the ear was negative. Wassermann reaction negative. Examination showed that the escaped fluid had the same composition as that of a normal cerebrospinal fluid. Eye examination had no bearing on the symptoms. Complete neurologic examination showed no evidence of organic disease of the brain.

Conclusion.—“Patient became so depressed and melancholic that it was deemed advisable to send her to the hospital for a rest cure. After being in the hospital two and a half months the flow of fluid from her left nostril entirely subsided; two weeks following the cessation of the flow she was suddenly taken with a chill and a rise of temperature of 104° , associated with this was numbness of the entire body and severe headache. Two days later she went into a semicomatose state with alternate periods of lucidity. The following day she developed a facial paralysis and tremors of the entire body. Two days following she developed auditory nerve deafness and three weeks later died of acute encephalitis.

DISCUSSION.

DR. FETTEROLF: To my mind, this was one of the most notable presentations to which I have ever listened, for carefulness of study and for accuracy and clearness of presentation it is particularly notable.

DR. WARREN B. DAVIS stated that he had been asked to see the patient while in the St. Agnes Hospital because of her pain, which had developed in the left ear. He stated that examination of the ear was negative. There was one point, which he stated Dr. Loftus had not mentioned, and that was the possibility of a fracture having been made at the time of a submucous resection, which had been performed some time before, but was unable to determine whether this was the case. The upper part of the resection seemed to have been done fairly high. There was a firmness of the septum where Dr. Loftus said there was still a ridge. There was also a slight palsy on the left side at the time of his examination.

DR. GEORGE B. WOOD inquired if a postmortem had been secured.

DR. LOFTUS stated that unfortunately, due to the absence of a pathologist, a postmortem had not been done. He had been given the privilege, but the proper person was not around at the desired time. Regarding the escape of cerebrospinal fluid from the ear: In reviewing the literature, he found that Escat reported a case of cerebrospinal fluid from the left ear following a submucous resection. No other cases were found in the literature.

**A Series of Colored Paintings Illustrating in Progression the
Pathologic Conditions of the Membrana Tympani.**

BY JAMES H. MENDAL, M. D.

(By invitation.)

The author showed in succession painted illustrations 200 times larger than the normal size of the drumhead, taking up in succession the normal ear drum and the various pathologic conditions which are found. The plates represented an enormous amount of work and careful study made by the author. The minute details that were brought out and explained were both interesting and instructive to all members present.

INDEX OF AUTHORS.

- BABBITT, J. A., 879.
Berry, G., 807.
Barany, R., 595.
Bigelow, I., 641.
Boot, G. W., 1241, 1252.
Bozer, H. E., 277.
Brown, L. E., 367.
Burton, F. A., 97.
Butler, R., 1259.
- CAVANAUGH, J. A., 1251.
Charlton, C. C., 485.
Costa, O. G., 923.
- DEAN, L. W., 285.
Demetriades, T., 894.
Dennis, F. L., 160.
Downey, J. W., 923.
Dutrow, H. V., 398.
- EVES, C. C., 904.
- FENTON, R. A., 67, 526, 820.
Fields, S. O., 583.
Frank, I., 768.
Fraser, J. S., 953.
- GERBER, I., 641.
Graham, H. B., 886.
Grichter, B. B., 860-936.
Grove, W. E., 855-926.
- HASKIN, W. H., 487.
Hayden, A. A., 622.
Holinger, 928.
Hubbard, T., 298.
Hubert, L., 749.
Hurd, L. M., 394.
- ISRAEL, S., 504.
Ivy, R. H., 1197.
- JARVIS, D. C., 405.
Johnston, W., 201.
- KEIPER, G. F., 236.
Kelly, I. D., 1053.
Kenyon, E. L., 606.
Kranz, F. W., 545.
Kully, B. M., 437.
- LEBENSOHN, J. E., 1128, 1281.
Lewis, F. O., 305.
Lewis, J. D., 321.
Lewy, A., 1086.
- Loeb, H. W., 1103.
Long, C. H., 635, 1351.
Looper, E. A., 417.
Lyons, H. R., 457.
- MACKENZIE, G. W., 427, 847, 940.
McCullagh, S., 1083.
Maybaum, J. L., 273, 875.
Minton, J. P., 45, 85, 316.
Morris, A. G., 864.
Mosher, H. P., 1.
- ODENEAL, T. H., 561.
- PACIENZA, F. A., 923.
Patton, J. M., 58.
Pearlman, S. J., 860-936.
Phillips, W., 780.
Pierce, N. H., 616.
Pohlman, A. G., 545.
Pratt, J. A., 211.
Prenn, J., 413.
Proetz, A. W., 223, 231, 233.
- RICHARDS, L. G., 1145.
Ridpath, R. F., 464.
Ross, E. L., 1229-1253.
Roy, Dunbar, 129.
Roy, J. N., 519, 554.
Rundstrom, A., 607.
- SALINGER, S., 934.
Sauer, W. E., 25.
Sautter, C. M., 118.
Shambaugh, G. E., 443, 636.
Skillern, R. H., 139.
Sluder, G., 1108.
Smith, S. M., 1203.
Sonnenschein, 45, 85, 316, 827.
Spencer, F. R., 838.
Stein, O. J., 1214.
- VAN DER HOEVE, J., 571.
Von Colditz, 149.
- WARING, J. B. H., 913.
Webber, R., 1246.
Weinberger, N. S., 387.
Weller, C. V., 687.
Westlake, S. B., 739.
Whitman, J. D., 474.
Willcutt, G. H., 1121.
Wood, G. B., 1259.
- ZEBROWSKI, 453, 1224.

INDEX OF TITLES.

- ABSCESS brain, Zebrowski, 1224.
 Abscess lung, J. Prenn, 413.
 Abscess vitreous, S. Salinger, 934.
 Adenoids, I. D. Kelly, 1053.
 Alcohol injection, R. Sonnenschein, 827.
 Anesthesia, E. L. Ross, 1229, 1253.
 Anesthesia local, C. C. Evê's, 904.
 Aphonia, J. A. Babbitt, 879.
 Arthritis larynx, S. O. Fields, 583.
 Atlas dislocation, G. F. Keiper, 236.
 Atresia choanae, J. E. Lebensohn, 1128, 1281.
 BRONCHOSCOPY, T. Hubbard, 298.
 Bronchoscopy, B. M. Kully, 437.
 CALCULUS salivary, A. W. Proetz, 233.
 Cancer nose, R. Butler, 1259.
 Cancer throat, R. Butler, 1259.
 Carotid gland, G. W. Boot, 1241, 1252.
 Cholesteatoma, S. M. Smith, 1203.
 Choanae atresia, J. S. Lebensohn, 1128, 1281.
 Cleft palate, C. H. Long, 635.
 Cochlea-palpebral reflex, T. Demetriades, 894.
 Codein poisoning, A. W. Proetz, 231.
 Crista ampularis, G. E. Shambaugh, 443, 636.
 Cyst tonsil, A. W. Proetz, 223.
 DACRYOCYSTOTOMY, R. A. Fenton, 67; H. P. Mosher, 1; W. E. Sauer, 25.
 Deafness, W. Phillips, 780.
 Deafness mumps, G. H. Willcutt, 1121.
 Deformities nasal, J. D. Lewis, 321.
 Deformities nose external, S. Israel, 504.
 Dental relations, W. H. Haskin, 487.
 Diaphragm hernia, L. G. Richards, 1145.
 Diathermy, A. Lewy, 1086.
 Diphtheria, R. Webber, 1246.
 Diplegia, G. F. Keiper, 236.
 Dust inhalation, D. C. Jarvis, 408.
 EIGHTH nerve paralysis, G. F. Keiper, 236.
 Empyema maxillary sinus, J. L. Maybaum, 273.
 Epithelioma esophagus, S. McCullagh, 1083.
 Esophagoscopy, T. Hubbard, 298.
 Esophagus epithelioma, S. McCullagh, 1083.
 Esophagus foreign body, J. A. Cavanaugh, 1251.
 Ethmoid cell, S. B. Westlake, 739.
 Eustachian tube, N. H. Pierce, 616.
 Exostosis aural, F. A. Burton, 97.
 Eye and ear relation, J. van der Hoeve, 571.
 FACIAL diplegia, G. F. Keiper, 236.
 Fatalities operation, H. W. Loeb, 1103.
 Fifth nerve paralysis, G. F. Keiper, 236.
 Fistula parotid, W. E. Grove, 855, 926.
 Foreign body, J. A. Cavanaugh, 1251.
 Foreign body, H. B. Graham, 886.
 Fracture nasal, J. Frank, 768.
 Frontal sinus radical operation, F. O. Lewis, 305.
 GASSERIAN ganglion, H. R. Lyons, 457.
 Growths nasal, J. A. Pratt, 211.
 HARE lip, C. H. Long, 635.
 Hay fever, O. J. Stein, 1214.
 Hearing acuity, A. G. Pohlman and F. W. Kranz, 545.
 Hearing aid, G. Berry, 807.
 Hemimacroglossia, J. N. Roy, 519.
 INHALATION dust, D. C. Jarvis, 405.
 Intranasal suture, W. Johnston, 201.
 JAW tumors, J. D. Whitman, 474.
 LABYRINTH, J. S. Fraser, 953.
 Labyrinth, J. Holinger, 928.
 Labyrinthitis syphilitic, G. W. Mackenzie, 847-940.

- Lacrimal sac anatomy, J. M. Patton, 58.
 Lacrimal sac operation, R. A. Fenton, 67; H. P. Mosher, 1; W. E. Sauer, 25.
 Larynx arthritis, S. O. Fierds, 583.
 Larynx tuberculosis, F. R. Spencer, 838.
 Lung abscess, J. Prenn, 413.
- MALNUTRITION** children, T. H. Odeneal, 561.
 Mastoid diphtheria, R. Webber, 1246.
 Mastoiditis, D. Ray, 129.
 Mastoid operation, J. W. Downey, F. A. Pacienza, O. G. Costa, 923.
 Mastoid operation, J. L. Maybaum, 875.
 Mastoid operation, radical, C. M. Sautter, 118.
 Mastoid process, N. Bigelow and J. Gerber, 641.
 Mastoiditis, R. A. Fenton, 820.
 Mastoiditis, T. H. Odeneal, 561.
 Maxillary sinus empyema, J. L. Maybaum, 273.
 Maxillary sinusitis, H. V. Duntrow, 398.
 Maxillary sinus, R. H. Ivy, 1197.
 Meningitis otogenic, J. W. Downey, F. A. Pacienza, O. G. Costa, 923.
 Middle ear diphtheria, R. Webber, 1246.
 Mosher-Toti operation, Mosher, 1.
 Mumps deafness, G. H. Willcutt, 1121.
- NASAL** fracture, I. Frank, 768.
 Nasal growths, J. A. Pratt, 211.
 Nasal reflex, R. F. Ridpath, 464.
 Nasal sinuses, R. H. Skillern, 139.
 Neurolabyrinthitis, syphilitic, G. W. Mackenzie, 847-940.
 Nose cancer, R. Butler, 1259.
 Nose external deformity, S. Israel, 504.
 Nose obstruction, C. H. Long, 1251.
 Nose polyp, G. B. Wood, 1259.
 Nystagmus, G. W. Mackenzie, 427.
- OPERATION** fatalities, H. W. Loeb, 1103.
 Optic nerve, S. B. Westlake, 739.
 Otitis media, H. R. Lyons, 457.
 Otitis media, T. H. Odeneal, 561.
 Otitis media, G. T. von Colditz, 149.
 Otitis media suppurative, H. E. Bozer, 277.
 Otogenic meningitis, J. W. Downey, F. A. Pacienza, O. G. Costa, 923.
 Otolaryngology, A. A. Hayden, 622.
 Otoneurology, R. Barany, 595.
 Otoscopy, A. Zebrowski, 453.
 Ozena, A. Rundstrom, 607.
- PAIN** mechanism, L. Hubert, 749.
 Palate cleft, C. H. Long, 635.
 Paralysis eighth nerve, G. F. Keiper, 236.
 Paralysis fifth nerve, G. F. Keiper, 236.
 Paralysis third nerve, G. F. Keiper, 236.
 Paranasal sinus, T. H. Odeneal, 561; R. H. Skillern, 139.
 Paranasal sinus disease, L. W. Dean, 285.
 Parotid fistula, W. E. Grove, 855-926.
 Perforation septum nasal, J. N. Roy, 554.
 Phlebothrombosis sinus, C. C. Charlton, 485.
 Polyarthritides, D. Roy, 129.
 Polyp nose, G. B. Wood, 1259.
- RADIOGRAM** mastoid, N. Bigelow and I. Gerber, 641.
 Radium, R. Butler, 1259.
 Reflex nasal, R. F. Ridpath, 464.
 Resection submucous, N. S. Weinberger, 387.
 Resonators Schaefer's, R. Sonnenschein and J. P. Minton, 45-316.
 Rinne test, R. Sonnenschein and J. P. Minton, 85, 316.
- SAC** lacrimal anatomy, J. M. Patton, 58.
 Sac lacrimal operation, R. A. Fenton, 67; H. P. Mosher, 1; W. E. Sauer, 25.
 Salivary calculus, A. W. Proetz, 233.
 Schaefer's resonators, R. Sonnenschein and J. P. Minton, 45, 316.
 Septum nasal perforation, J. N. Roy, 554.
 Sinus frontal, radical operation, F. O. Lewis, 305.
 Sinus infection, L. M. Hurd, 394.
 Sinus infection, E. A. Looper, 417.
 Sinus intracranial phlebothrombosis, C. C. Charlton, 485.

- Sinus maxillary, R. H. Ivy, 1197.
Sinus maxillary empyema, J. L. Maybaum, 273.
Sinus paranasal, T. H. Odeneal, 561. R. H. Skillern, 139.
Sinus paranasal disease, L. W. Dean, 285.
Sinus thrombosis nasal, S. Salinger, 934.
Sinusitis, R. A. Fenton, 526.
Sinusitis maxillary, H. V. Duntrow, 398.
Sinusitis paranasal, M. F. Arbuckle, 608.
Sound reinforcement, R. Sonnenschein and J. P. Minton, 45, 316.
Sphenoid fissure, G. Sluder, 1108.
Submucous resection, N. S. Weinberger, 387.
Sutures intranasal, W. Johnston, 201.
Swimming sinusitis, R. A. Fenton, 526.
THIRD nerve paralysis, G. F. Keiper, 236.
Throat cancer, R. Butler, 1259.
Thrombosis sinus, C. C. Charlton, 485.
Thrombosis sinus nasal, S. Salinger, 934.
Thyrotroxin, L. E. Brown, 367.
Tonsil, A. G. Morris, 864.
Tonsil bone, C. V. Weller, 687.
Tonsil cartilage, C. V. Weller, 687.
Tonsil cyst, A. W. Proetz, 223.
Tonsil infection, L. E. Brown, 367.
Tonsillectomy, B. B. Grichtler and S. J. Pearlman, 860-936.
Tonsillectomy, E. L. Ross, 1229, 1253.
Tonsillectomy, R. Sonnenschein, 827.
Tonsillectomy, J. B. H. Waring, 913.
Tuberculin, G. T. von Colditz, 149.
Tuberculosis larynx, F. R. Spencer, 838.
VERTIGO persistent, J. L. Maybaum, 875.
Vestibule tests, F. L. Dennis, 160.
Vitreous abscess, S. Salinger, 934.
Voice impaired, E. L. Kenyon, 606.

(19)

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and laryngology

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